INDUSTRIAL HYGIENE MANUAL

ATSC No. 25-0-1

INDUSTRIAL TOXIC AGENTS

CHEMICAL FORMULA AND SYNONYMS

PRINCIPAL PROPERTIES

PRINCIPAL INDUSTRIAL USES

POISONING—SYSTEMIC

MAXIMUM PERMISSIBLE CONCENTRATIONS OF ATMOSPHERIC CONTAMINANTS

Office of the Surgeon

HEADQUARTERS AIR TECHNICAL SERVICE COMMAND

WRIGHT FIELD, DAYTON, OHIO

FOREWORD

This manual was compiled from recently published articles and texts concerning Industrial Hygiene subjects. It is suggested that the information presented be used as far as possible under the existing facilities available at the respective depots. At the end of each related group will be found a list of references to articles or books that have been especially drawn upon, and it is the desire of this office to acknowledge our obligations to these sources as well as to refer the reader to them for further study of particular problems encountered in the various depots.

CONTENTS

MAXIMUM PERMISSIBLE CONCENTRATIONS OF ATMOSPHERIC CONTAMINANTS AS RECOMMENDED BY VARIOUS STATE INDUSTRIAL HYGIENE UNITS.

Substance	Concentration
Acetone	200 p.p.m.
Aliphatic acetates	500 p.p.m.
Ammonia	100 p.p.m.
Amyl acetate (n)	500 p.p.m.
	400 p.p.m.
Aniline	5 p.p.m.
Arsenic trioxide	0.5 mg./cu.m.
Arsine	1 p.p.m.
Benzene	100 p.p.m.
	75—100 p.p.m.
	75 p.p.m.
Bromine	1 p.p.m.
Butanol	100 p.p.m.
Butyl acetate	500 p.p.m.
	400 p.p.m.
Cadmium	0.1 mg./cu.m.
Carbon dioxide	5500 p.p.m.
Carbon disulfide	20 p.p.m.
	15 p.p.m.
Carbon monoxide	100 p.p.m.
Carbon tetrachloride	100 p.p.m.
Chlorine	75 p.p.m.
	1 p.p.m.
Chlorodiphenyl	1.0 mg./cu.m.
Chloroform	100 p.p.m.
Chloronaphthalenes	1—5 mg./cu.m.
" (above "tri")	1.0 mg./cu.m.
" ("tri")	5.0 mg./cu.m.
" (penta)	0.5 mg./cu.m.
Chromium (hexavalent)	0.1 mg./cu.m.
Chromium	0.1 mg./cu.m.
Dichlorobenzene (para)	75 p.p.m.
Dichloroethylene (trans)	100 p.p.m.
Dichloroethyl ether	15 p.p.m.
Ethanol	250 p.p.m.
Ethyl bromide	1700 p.p.m.
Ethyl chloride	2000 p.p.m.
Ethyl ether	400 p.p.m.

Substance	Concentration
Ethylene dichloride	100 p.p.m.
Formaldehyde	20 p.p.m.
Gasoline	1000 p.p.m.
Hydrogen chloride	10 p.p.m.
Hydrogen cyanide	20 p.p.m.
Hydrogen fluoride	3 p.p.m.
Hydrogen sulfide	50 p.p.m.
	20 p.p.m.
Iron Oxide fume (Fe_2O_3)	15 mg./cu.m.
Lead	0.15 mg./cu.m.
Magnesium oxide fume	15 mg./cu.m.
Manganese	50 mg./cu.m.
	6 mg./cu.m.
	5 mg./cu.m.
Mercury	0.1-0.2 mg./cu.m.
	0.15 mg./cu.m.
	0.1 mg./cu.m.
Methanol	200 p.p.m.
	100 p.p.m.
Methyl bromide	50 p.p.m.
Methyl chloride	500 p.p.m.
Monochlorobenzene	75 p.p.m.
Naphtha	5000 p.p.m.
Nitrobenzene	5 p.p.m.
	1 p.p.m.
Nitrogen oxides	29-70 p.p.m.
	40 p.p.m.
	10 p.p.m.
Ozone	1 p.p.m.
	0.1 p.p.m.
Perchlorethylene	100 p.p.m.
Petroleum vapors	1000 p.p.m.
Phosgene	1 p.p.m.
Phosphine	2 p.p.m.
Phosphorus trichloride	0.7 p.p.m.
Sulfur dioxide	10 p.p.m.

Substance	Concentration
Tetrachloroethane	10 p.p.m.
Tetrachloroethylene	200 p.p.m.
Toluene	200 p.p.m.
	100 p.p.m.
Trichlorethylene	200 p.p.m.

Substance	Concentration
	100 p.p.m.
Turpentine	700 p.p.m.
	200 p.p.m.
Xylene	200 p.p.m.
	100 p.p.m.
Zinc oxide fume	15 mg/cu.m.

DUSTS

Millions of particles per cubic foot of air—light field count.

Substance	Concentration
Alundum	15
Asbestos	15
	5
Carborundum	15
Cement	15
Feldspar	10
Foundry	20
	15
	12
Granite	25
	10
Mica	50
	10
Nuisance	50
Pottery	4
Organic	50
Pyrophyllite talc	25
	10

Substance	Concentration
Silica (based on percentage of free silica in the dust)	
Count x%	5
10%	50
("low")	50
10%	10
25—35%	10
"medium"	20
"high"	5
over 75%	5
over 90%	5
Silicates	15
Slate	50
	15
Soapstone	50
Talc	50
	15
Total dust	50

ACETONE: RELATED SOLVENTS

Chemical Formula and Synonyms:

(Acetone) $CH_3CO\,CH_3$, dimethyl ketol; dimethyl ketone; propanone; pyroacetic ether; ketopropane; methylacetyl.
(Methyl ethyl ketone) $CH_3CO\,C_2H_5$, butanone; ethylmethyl ketone.
(Methyl propyl ketone, N.) CH_3-CO- $(CH_2)_2CH_3$ 2- pentanone.
(Methyl butyl ketone, N.) CH_3-CO-C_4H_9, hexanone-2.

A. **Principal Properties:**

1. Acetone
   a. Colorless liquid; fragrant, mint-like odor; inflammable. Sp. gr. 0.7799 at 15°C.
   b. Soluble in water, alcohol, ether, chloroform, most volatile oils. Wt. of vapor per liter, 2.47 gr.
2. Methyl ethyl ketone
   a. Colorless liquid; acetone-like odor; inflammable. Sp. gr. 0.805;
   b. Soluble in water, alcohol and ether.
3. Methyl propyl ketone, N.
   a. Colorless liquid. Sp. gr. 0.812 at 15/15°C.
   b. Very slightly soluble in water; soluble in alcohol and ether in all proportions.
4. Methyl butyl ketone, N.
   a. Colorless liquid. Sp. gr. 0.830;
   b. Very slightly soluble in water; soluble in all proportions in alcohol and ether.

B. **Principal Industrial Uses:**

1. Acetone
   a. Solvent in manufacture of smokeless powder, varnishes, lacquers, airplane dopes, indigo, dyes of the diphenylamine series; intermediates; isoprene, artificial leather, adhesive mixtures (from nitrocellulose), lubricants, artificial perfumes, pharmaceuticals (chloroform, iodoform, bromoform, sulfonals), plastics, rubber cements; extraction of fats and oils, extraction of tannins from nut galls, purification of paraffin; substitute for alkali in photographic developers; impregnating raw cotton when dyeing with aniline black by oxidation; absorbent for acetylene gas.
2. Methyl ethyl ketone
   a. Organic synthesis; manufacture of smokeless powder; solvent.

C. **Poisoning—Systemic:**

1. Modes of entry
   a. Inhalation.
2. Symptoms
   a. Acetone is one of the less toxic solvents used extensively in industrial processes. High concentrations cannot be inhaled for more than a few minutes on account of acute irritation of the throat and eyes. Animal experiments indicate that for brief exposures high concentrations of acetone vapors are more toxic than chloroform or carbon tetrachloride and only slightly less toxic than benzol. This relative toxicity, however, applies only in regard to acute poisoning. The symptoms of acute poisoning are loss of equilibrium and light narcosis and if exposure is continued deep narcosis follows. It is said that acetone also exerts a stimulating action on the respiratory center. Prolonged exposure to low concentrations of acetone vapor does not seem to cause dangerous results.
   Although the destructive action of acetone in the moderate concentrations usually encountered in industrial processes does not appear to be of serious consequence, it should be emphasized that injury may result from solvent mixtures in which acetone is accompanied by some of the more dangerous solvents.

SELECTED REFERENCES

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, p. 193.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, p. 156.

Jones, G. W., Harris, E. S., and Beattie, B. B.: Protection of Equipment Containing Explosive Acetone—Air Mixtures by the Use of Deaphrams. U. S. Bur. Mines, Tech. Paper 553, p. 24, 1933.

Jones, G. W., Harris, E. S., and Miller, W. E.: Explosive Properties of Acetone—Air Mixtures. U. S. Bur. Mines, Tech. Paper 544, p. 26, 1933.

Jones, G. W. and Klick, J. R.: Inflammability of Mixtures of Ethyl Alcohol, Benzene, Furfural, and Acetone. Indust. and Engin. Chem., Vol. 21, pp. 791-793, 1929.

Kamlet, Jonas: The Detection of Acetone and Acetoacetic Acid in Urine. J. Lab. Clin. Med., vol. 24, pp. 206-207, 1938.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 631-633.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, p. 664.

Morbid Disturbances Due to the Handling of Celluloid: Jour. Am. Med. Assn., vol. 79, no. 2, p. 146, July 8, 1922.

Ohio Department of Health, Acetone and Related Solvents, 1940.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 37-38.

Teiss, R., and Wagner, O.: A Method of Determining Traces of Acetone. Gigiena truda i teknika bezopasnosti, no. 1, pp. 63-67, 1935.

Usdina, J.: Determining Acetone in Air. Gigiena bezopasn. i. patolog. truda, no. 506, pp. 155-158, 1933.

ALCOHOLS: ACETATES OF THE ALIPHATIC

Chemical Formula and Synonyms:

(Methyl acetate) CH_3CO_2-CH_3.
(Ethyl acetate) CH_3-CO_2-CH_2-CH_3, acetic ether; aether aceticus; vinegar naphtha.
(Propyl acetate) CH_3CO_2-CH_2-C_2H_5.
(Butyl acetate) CH_3CO_2-C_4H_9.
(Amyl acetate) $CH_3CO_2C_5H_{11}$, amyl acetic ether; banana oil, isoamyl acetate; pear oil.

A Principal Properties:

1. Methyl acetate
   a. Colorless, volatile, inflammable liquid; fragrant odor. Sp. gr. 0.92438.
   b. Soluble in water, alcohol and ether.
2. Ethyl acetate
   a. Colorless, fragrant, inflammable liquid. Sp. gr. 0.9003.
   b. Soluble in chloroform, alcohol and ether; slightly soluble in water.
3. Propyl acetate
   a. Colorless liquid. Sp. gr. 0.886 at 24/4°C.
   b. Soluble in all proportions in alcohol and ether; slightly soluble in water.
4. Butyl acetate
   a. Limpid, colorless, inflammable liquid, fruity odor; Sp. gr. 0.872 at 21°C.
   b. Soluble in alcohol, ether and hydrocarbons; slightly soluble in water.
5. Amyl acetate
   a. Colorless liquid; pear or banana-like odor. Sp. gr. 0.880 at 12.50°C.
   b. Soluble in alcohol or ether; very slightly soluble in water.

B. Principal Industrial Uses:

1. Methyl acetate
   a. Solvent; extracts; perfumery; artificial leather; plastics; solvent for nitrocellulose and acetyl cellulose.
2. Ethyl acetate
   a. Medicine; solvent; organic synthesis; used in manufacture of flavoring; smokeless powders; artificial fruit essences, bonbons and confection; artificial leather; solvent for phosgene gas; artificial bristles and horsehair; nitrocellulose varnishes, lacquers and dopes; nitrocellulose plastics; pharmaceuticals; rayon.

3. Propyl acetate
   a. Solvent for cellulose nitrate; ester gum; sandaroc; colophony; manila; cumarone; mastic, etc.
4. Butyl acetate
   a. Solvent in production of lacquers, lacquer enamels, pyroxlin solutions, leather dope, airplane dope, perfumes, flavoring extracts; solvent for natural gums and synthetic resins.
5. Amyl acetate
   a. Preparation of flavoring compounds; solvent for nitrocellulose; in the preparation of lacquers and photographic and motion picture films; waterproofed varnishes; waterproofing compounds; bronzing liquids; metallic paints; perfumery; solvent for tannins; artificial leather; artificial pearls; solvent for camphor; the combustible substance in the standard lamp used in photometry; solvent in manufacture of celluloid and celluloid cements; rayon; dyeing, printing and finishing textile fabrics.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation.
   b. Ingestion.
2. Symptoms
   a. The toxic status of the acetates of the aliphatic alcohols is still somewhat unsettled. It is evident, however, that injury resulting from exposure to their vapors is considerably less severe than certain other widely used solvents such as benzol, toluol, carbon tetrachloride, methyl alcohol, and petroleum distillates.

   Methyl, ethyl, propyl, butyl, and amyl acetates are similar in most of their physiological characteristics with certain minor variations due to differences in volatility and other physical properties. All are characterized by irritation of the mucous membranes of the eyes and upper respiratory tract. There is often a dryness and burning of the throat with inclination to cough and itching and burning of the eyelids. Narcotic action has also been observed for all of these substances. The intensity of this action may vary from a feeling of slight weariness to acute narcosis and is dependent upon the composition and concentration of the vapors encountered. In actual industrial practice the effects are usually limited to irritation of the mucous membranes of the upper respiratory tract and a feeling of dopiness.

   The real danger in exposure to the acetates of the aliphatic alcohols which are almost universally found in lacquers lies in the possibility of a more serious exposure to vapors of methyl alcohol, ether, benzol, toluol, chlorinated hydrocarbons.
   And petroleum distillates. These substances may be used as lacquer diluents and their presence is disguised by strongly odorous acetate such as amyl acetate. It is not uncommon for workmen to suffer from symptoms of benzol poisoning which have been attributed to amyl acetate, a relatively harmless solvent in comparison to benzol.

D. Sanitary Corps Officer:

1. Control Measures.

SELECTED REFERENCES

Blina, Luciano Vernetti: Experimental Investigation of the Toxic Action of Acetic Acid Esters. Amyl Acetate. Med. del Lav., vol. 24, pp. 166-178, 1933.

Burke, W. J., and Goldwater, L. J.: Hazards Incidental to Industrial Uses of Nitrocellulose Lacquers. N. Y. State Dept. Labor, Ind. Bull., vol. 17, pp. 314-316, July 1938.

Cavallazza, Desiderio: Butyl Acetate and its Toxic Action. Rass. Med. Applicata Lavaro Ind., vol. 9, pp. 272-282, 1938.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 191-193.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, p. 157.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, p. 644.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 663-664.

Occupation and Health. International Labour Office, Geneva, 1934, p. 114.

Ohio Dept. of Health: Acetates of the Alipahatic Alcohols, 1940.

Ohnesorge, G.: On Poisoning by Zapon Lacquer. Deutsch. Med. Wchnschr., vol. 56, pp. 961-963, 1930.

Sharpe, N. C.: Amyl Acetate Poisoning. Pub. Health Jour., vol. 14, pp. 110-118, March, 1923, pp. 172-174, April 1923.

Undine, C. A.: Duco Poisoning: Report of Case. Minnesota Med., vol. 11, p. 703, Oct. 1928.

ALCOHOL: METHYL

Chemical Formula and Synonyms:

CH_3-OH Acetone alcohol; columbian spirits; colonial spirits; green wood spirits; manhattan spirits; methanol; methyl hydrate; methyl hydroxide; standard wood spirits; wood alcohol; wood naphtha; wood spirits.

A. **Principal Properties:**

1. Clear, colorless, mobile, volatile, inflammable liquid; poisonous. Sp. Gr. 0.7970 at 15°C. Soluble in water, alcohol, and ether. Weight per liter of vapor 1.33 g.

B. **Principal Industrial Uses:**

1. Manufacturing formaldehyde; organic synthesis; denaturing ethyl alcohol; general solvent; fuel; smokeless powders; paints, varnishes, paint removers; polishing and cleaning preparations; transparent and disinfecting soaps; dry cleaning; anti-freeze; fuel compositions used for heating and illuminating; perfumery.

C. **Poisoning—Systemic:**

1. Modes of entry.
   a. Inhalation.
   b. Ingestion.
   c. Absorption through the skin.
2. Symptoms.
   a. Locally, methyl alcohol is an irritant to the mucous membranes of eyes, nose, and respiratory system. The skin may be dry, inflamed, or eczematous. When absorbed, causes headache, weakness, vertigo, nausea and vomiting, dilated pupils, fogginess of vision, visual hallucinations, severe colic, gastric congestion with hemorrhagic diarrhea, rapid breathing followed by retardation, acceleration and slowing of the heart, disturbance of pulse, cold sweats, cyanosis, sighing, loss of reflexes and sensation, decreased temperature, nystagmus, sweating, delirium, convulsions, paralysis, coma, and may result in pneumonia. Diplopia may result from paralysis of external eye muscles; inflammation, neuritis, and atrophy of the optic nerves are common, resulting in blurred vision, then blindness, which is bilateral. Blindness may come on in a few hours or in a few days; in typical cases there is often transient improvement followed by complete and permanent blindness.
   b. Slow chronic poisoning gives no characteristic symptoms and may result in severe damage before recognized; however, the Germans believe that vague nervous symptoms, a sense of fatigue, and irritation of the mucous membranes should lead to suspicion of poisoning. McCord has shown that poisoning in animals takes place as readily through the skin and lungs as through the stomach and intestines.
   c. Elimination: Slowly excreted in the urine and expired air, some into the stomach, and part is slowly oxidized with the formation of formic acid which will reduce Fehling's solution and may suggest a false diagnosis of diabetes.
3. Toxic Concentrations.
   a. It has been found that concentrations of 20-25 parts per million were not toxic.
4. Pathology.
   a. Changes consisted of marked edema, hyperemia, and necrosis of the stomach, intestine, liver, brain, and retina.

D. **Sanitary Corps Officer:**

1. Control of ventilation.
2. Proper storage and sound plumbing tubes leading it off.
3. Pointing out dangers to workmen exposed and its danger as a beverage.

SELECTED REFERENCES

Albaugh, R. P.: Wood Alcohol Poisoning. Med. Med., vol. 1, no. 4, p. 300, Aug. 1919.

Bertarelli, E.: The Problem of the Toxicity of Methyl Alcohol. Ann. d'ig., vol. 42, pp. 665-676, Oct. 1932.

Flury, F., and Wirth, W.: Methyl Alcohol and Toxic Methyl Compounds. Arch. f. Gewerbepathol., vol. 7, pp. 221-226, 1936.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 186-190.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 166-169.

Isaacs, Raphael.: Acute Methyl Alcohol Poisoning. Jour. Am. Med. Assn., vol. 75, no. 11, pp. 718-721, Sept. 11, 1920.

Keeser, E.: Cause of Toxicity of Methyl Alcohol. Deutsch. Med. Wchnschr., vol. 57, p. 398, March 6, 1931.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, p. 654.

Koelsch, F.: Hazards in Working with Denatured Alcohol. Burnisher's Exzema. Zentralbl. f. Gewerbehyg., vol. 9, no. 9, pp. 203-207, Sept. 1921.

McCord, C. P.: Toxicity of Methyl Alcohol (Methanol) Following Skin Absorption and Inhalation. A Progress Report. Indust. and Engin. Chem., vol. 23, pp. 931-936, Aug. 1931.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 613-631.

Occupation and Health, International Labour Office, Geneva, 1934, pp. 233-238.

Ohio Department of Health, Methyl Alcohol, 1940.

Pohl, J.: An Investigation of the Detoxication of Methyl Alcohol. Arch. f. Exper. Path. u. Pharmakol., vol. 83, no. ¾, pp. 204-220, April 1918.

Sayers, R. R., and Yant, W. P.: Observations and Notes on the Effect of Methanol Antifreeze on Health. U. S. Bur. Mines, Information Circ. No. 6415, p. 5, Dec. 1930, (Multigraphed).

Snell, F., and Snell, D.: Colorimetric Methods of Analyses. D. Van Nostrand Co., Inc., Publishers, New York, 1937, pp. 17-20.

Trumper, M.: Antifreeze Methanol Hazard. Abst. as Follows from Internat. Clin., vol. 1, pp. 85-89, 1931, in Chem. Abstr., vol. 25, p. 2783, June 10, 1931.

U. S. Bur. Labor Statis.: Recommendations Concerning the Manufacture and Use of Wood Alcohol. Month. Labor Rev., vol. 10, no. 2, pp. 197-201, Feb. 1920.

Yant, W. P., and Schrenk, H. H., and Sayers, R. R.: Methanol Antifreeze and Methanol Poisoning. Indust. and Engin. Chem., vol. 23, pp. 551-555, May 1931.

Ziegler, S. Lewis: The Ocular Menace of Wood Alcohol Poisoning. Jour. Am. Med. Assn., vol. 77, no. 15, pp. 1160-1166, Oct. 8, 1921.

ALDEHYDES

Chemical Formula and Synonyms:

(Formaldehyde) HCHO: formalin; formalith; formic aldehyde; formol; oxymethylene.
(Acetaldehyde) CH_3CHO; ethyl aldehyde; acetic aldehyde.
(Acrolein) $CH_2{=}CH\text{-}CHO$: acraldehyde; acrylic aldehyde; allyl aldehyde; allylic aldehyde; propenal.
(Crotonaldehyde) $CH_3CH{=}CHCHO$.
(Furfural) $C_4H_3O\text{-}CH{=}O$; furfuraldehyde; furfurane carboxylic aldehyde; furfurol; furol; pyromucic aldehyde.

A. Principal Properties:

1. Formaldehyde.
   a. Gas; suffocating pungent odor; poisonous; Sp. gr. 0.815; m.p. –192°C.; b.p. –21°C. Miscible with water and alcohol. Forms an aqueous solution of formaldehyde gas. Weight per liter 1.25 gr.

2. Acetaldehyde.
   a. Colorless, light, inflammable liquid; pungent, fruity odor. Sp. gr. 0.783 at 18/4°C.; m.p. –123.5°C.; b.p. 20.2°C. Miscible with water, alcohol, ether, benzene, gasoline, solvent naphtha, toluene, xylene, turpentine, acetone.

3. Acrolein.
   a. Colorless or yellowish liquid, inflammable; disagreeable choking odor; violent action on the eyes; poisonous; Sp. gr. 0.8410 at 20°C.; m.p. –87.7°C.; b.p. 52.4°C. Soluble in water, alcohol and ether. Weight per liter, 2.3 gm.

4. Crotonaldehyde.
   a. Water-white mobile, inflammable liquid; pungent, suffocating odor; turns to pale yellow color in contact with light and air. An effective lachrymator. Sp. gr. 0.8567 at 15.6/4°C.; m.p. –69°C.; b.p. 102°C.; slightly soluble in water; miscible in all proportions with alcohol, ether, benzene, toluene, kerosene, gasoline, solvent-naphtha.

5. Furfural.

a. Colorless, mobile liquid when very pure; changes to reddish-brown upon exposure to light and air. Penetrating odor somewhat similar to benzaldehyde. Furfural forms condensation products with many types of compounds, (phenol, amines, urea, etc.) m.p. –36.5°C.; b.p. 161.7°C.; sp. gr. 1.1598 at 20/4°C.; heat of vaporization 107.5 calories; flash point 55.57°C.; refractive index 1.5260. Soluble in alcohol, ether and benzol; 8.3% soluble in water at 20°C. Weight per liter of vapor, 4.00 gr.

Principal Industrial Uses:

1. Formaldehyde.

a. Chemicals, (formic acid, formals from alcohol, urea, hydrosulfite derivatives, hexamethylenetetramine, acetaldehyde, paraformaldehyde); dyes (triphenylmethane dyes, anthracene dyes, quinoline dyes, acridine); explosives; medicine (disinfectant, germicide); embalming fluids; pharmaceuticals (deodorizing and rendering tasteless products from sulfonated mineral oils, making therapeutic preparations from wood tar distillation oils, applications for wounds, insect stings, etc., various preparations): insecticide; food (preserving milk, treatment of frozen meat on ships, curing foods), photography; plastics (phenol condensation products of various properties, condensation of cresol and other organic substances, bonelike articles from galalith, condensation agent in making cyclohexanone resins, securing insolubility and stability in casein preparations); brewing; distilled liquors; inks; leather (giving body to the surface of leather in tanning, preserving and stiffening the grain of hides); waterproofing straw hats; fixing hair on fur skins; preservative for botanical, zoological and bacteriological purposes; waterproofing paper by fixing gelatin or glue impregnated therein; disinfectant soaps; hardening and preservative agent in treatment of starches and starch preparations; preserving sugar cane syrups and juices; rayon; textile processes; silvering mirrors; metallurgy (reducing agent in recovery of gold and silver): rubber (preservative, coagulant).

2. Acetaldehyde.

a. Manufacture of chemicals, dyes, intermediates; yeast albumin; phenol condensation products; synthetic rubber, disinfectants; silvering mirrors; hardening dry gelatin films for photography.

3. Acrolein.

a. Organic synthesis.

4. Crotonaldehyde.

a. Solvent for varnishes, uncured synthetic resins, vegetable and minerals oils, rosin, shellac, wood distillation resin; solvent (on heating) for fats, waxes and rubber.

5. Furfural.

a. Synthetic resins; purification of organic materials by selective solvent action; nitrocellulose solvent; photoengraving processes; preservative; furoates and derivatives.

C. **Poisoning—Systemic:**

1. Modes of entry.

a. Chiefly by inhalation of fumes and vapors.

Symptoms.

a. General Considerations;
Aldehydes as a class are irritating to the skin and mucous membranes. The irritation is so marked that the exposed individual usually leaves the source of contamination before serious injury is effected. Aldehydes also have anesthetic properties, but this action is largely masked by the more intense irritant effect.

2. Formaldehyde.

a. The powerful irritant action of formaldehyde upon the mucous membranes is due to its forming an irreversible combination with the proteins of the surface cells. Irritation due to formaldehyde is most evident in the conjunctiva and mucous membranes of the upper respiratory system. Ingestion, which is usually accidental, results in severe irritation of the gastro-intestinal tract with nausea and vomiting.

Some observers have stated that the absorption of small quantities of formaldehyde over a period of time has a cumulative effect analagous to that of wood alcohol. This contention, however, has not been positively established. Exposure of an individual whose skin previously had a normal resistance to the action of formaldehyde may become acutely sensitive to its effect. Some authorities contend that a substantial amount of obscure occupational dermatitis may owe its origin to small quantities of formaldehyde.

3. Acetaldehyde.
   a. Inhalation of acetaldehyde vapors causes irritation of the mucous membranes of the eyes and upper respiratory tract. Strong concentrations cause a sensation of coughing, acceleration of the heart, night sweats, narcotic effects and methemoglobinuria.

4. Acrolein.
   a. Acrolein is encountered as a troublesome by-product when fats or vegetable oils are subjected to heat. Its fumes are heavy, thick, and very irritating and give rise to an instinctive impulse to hold the breath and keep it out of the lungs. Its effect is primarily on the upper respiratory tract in the low concentrations ordinarily inhaled but it will induce edema of the lungs in animals which are subjected to high concentrations. Prolonged exposure to low concentrations causes catarrhal inflammation in both the pharynx and larynx. The toxic concentrations of acrolein are about the same as those of phosgene and it has been tested as a possibility for chemical warfare.

5. Furfural.
   a. Furfural is locally corrosive and extremely irritating to the skin and mucous membranes. It is also believed to have a narcotic action. Its toxicity is said to be one-third that of formaldehyde.

SELECTED REFERENCES

Gardner, H. A.: Physiological Effects of Vapors from a Few Solvents Used in Paints, Varnishes and Lacquers. Paint Manufactures' Assn., Scientific Section, Circ. No. 250, pp. 89-149, 1925.

Hambersin, J. M.: The Determination of Traces of Formaldehyde. Bull. Soc. Chim. Belg., vol. 46, pp. 519-524, 1937.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 197-201, 227.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 129, 133, 155.

Hetzel, K. W.: Formalin as a Disinfectant for Gas Maske Equipment and the Most Convenient Method of Use. Draeger-Hefte, no. 174, pp. 2654-2665, 1934.

Jones, G. W., and Klick, J. R.: Inflammability of Mixtures of Ethyl Alcohol, Benzene, Furfural, and Acetone. Indust. and Engin. Chem., vol. 21, pp. 791-793, 1929.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, p. 643.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 36, 48, 806-810.

Ohio Department of Health, Aldehydes, 1940.

ANILINE: RELATED COMPOUNDS

A. Principal Properties:

1. Colorless oily liquid; rapidly becomes brown on exposure to air and light. Sp. Gravity 1.0235; soluble in alcohol and ether; slightly soluble in water.
2. Other compounds—Diethylaniline, Dimethylaniline.

B. Principal Industrial Uses:

1. Dyes; intermediates; chemicals (aniline salts, etc.); dyeing and calico printing; vulcanization accelator; germicide; paint and varnish; perfumery.

C. Poisoning—Systemic:

1. Modes of entry.
   a. Inhalation of fumes.
   b. Ingestion.
   c. Absorption through the skin. Inhalation and absorption through the skin by direct contact is the most frequent type of industrial poisoning.
2. Symptoms.
   a. Acute. There is sudden prostration; cold, pale skin; blue lips; diminuation of sensibility. In the mild cases of acute poisoning there is pallor of skin with cyanosis, vertigo, unsteady gait, slow labored speech, irritability.
   b. Chronic. Anemia, slowing of the pulse, disorders of digestion, such as anorexia, vomiting, and diarrhea are encountered. Nervous symptoms such as general debility, headache, ringing in the ears, vertigo and insomnia are evident.
3. Pathology.
   a. Aniline exercises its toxic effects chiefly on the blood and nervous systems. It changes the oxyhemoglobin of the red cells into methemoglobin, a transformation which is attended by marked cyanosis and a progressive diminuation in the power of the blood to absorb oxygen. Although the symptoms of internal asphyxia are detectable in the central nervous system, not all nervous symptoms are due to the secondary action of the hemoglobin transformation. Aniline produces, in addition, an early narcotic effect as well as other central nervous system disturbances before the oxygen carrying capacity of the blood is sensibly diminished.
   b. The occurrence of tumors, both malignant and benign, have been frequently reported among workers in aniline and its derivatives. A wide variety of compounds may give rise to bladder tumors, and medical authorities do not all agree that aniline alone, unaccompanied by other chemicals, is the cause of such tumors.
4. Laboratory Procedure.
   a. The presence of methemoglobin, Heinz's corpuscles in the blood, and of p-amidophenol in the urine are indicative of exposure to aniline or its derivatives.

D. Sanitary Corps Officer:

1. Active supervision of control measures where aniline or aniline derivatives are being used.

SELECTED REFERENCES

Aiello, G.: The Pathology of Aniline Poisoning. Med. du Travail, pp. 294-296, July 1930.

Albaugh, R. P.: Anilin Poisoning—Its Diagnosis and Treatment. Mod. Med., vol. 1, no. 5, pp. 398-399, Sept. 1919.

Alcayaga, L. E., and Bercovich, A.: Acute Poisoning Caused by a Tincture for Shoe Dyeing. Semana Med., vol. 36, pp. 642-648, 1929.

Arneth and Albacht.: Qualitative Blood Changes in Poisoning by Carbon Monoxide, Lysol, and Aniline Oil. Zentralbl. f. Gewerbehyg., N.S., vol. 4, pp. 225-228, July 1927.

Berenblum, I., and Bonser, G. M.: Experimental Investigation of "Aniline Cancer." Jour. Ind. Hygiene, vol. 19, no. 2, p. 86, Feb. 1937.

Berenblum, I.: Aniline Cancer. Cancer Rev., vol. 7, pp. 337-55, 1932.

Bonzanigo, A.: Industrial Poisoning with Aniline and Similar Substances. Deutsch. Ztschr. f. d. Ges. Gerichtl. Med., vol. 16, pp. 242-245, 1931.

Buttner, W.: Occupational Cancer in Aniline Dye Workers as Observed in the Surgical Clinic of Heidelberg University. Ztschr. f. Krebsforsch., vol. 34, p. 605, 1931.

Buzzi, A., Costa, A. J., and Derqui, M. N.: Hand and Finger Injuries Caused by Aniline Dye Pencils. Semama Med., vol. 36, pp. 1064-1068, 1929.

Castrovilli, Giuseppe: Acute Intoxication with Aniline Oil. Rass. Med. Applicata Laboro Ind., vol. 9, pp. 107-115, 1938.

Cathala, J., Hazard, R., Maschas, H., and Jequier, R.: Acute Poisoning by Ingestion of Aniline. Ann. Med., vol. 43, pp. 187-194, 1938.

Cronin, H. J.: Aniline Poisoning in the Rubber Industry. Boston Med. and Surg. Jour., vol. 191, p. 683, Oct. 9, 1924.

Davis, Paul A., M. D.: Aniline Poisoning in the Rubber Industry. Jour. Ind. Hygiene, vol. 3, no. 2, pp. 57-61, June 1921.

Davis, P. A.: Toxic Substances in the Rubber Industry. III. Aniline. Rubber Age., vol. 25, pp. 611-612, 1929.

de Balsac, F. H., Agasse-Lafont, E., and Feil, A.: An Inquiry into the Chronic Occupational Anilism of the Imitation Fur Industry. Jour. Med. Franc., vol. 17, pp. 377-380, Nov. 1928.

de Balsac, F. H.: Occupational Anilism at Certain Processes of Pharmaceutic Chemistry. Med. du travail, pp. 285-290, July 1930.

Ferguson, R. S., Gehrmann, G. H., Gay, D. M., Anderson, L. W., and Washburn, V. D.: Symposium on Aniline Tumors of the Bladder: Introduction. Jour. Urol., vol. 31, pp. 121-171, Feb. 1934.

Gley, P.: The Action of Aniline Derivatives on the Blood. Bull. Acad. Med., vol. 118, pp. 377-82, 1937.

Hamilton, Alice, M. D.: A Discussion of the Etiology of So-called Aniline Tumors of the Bladder. Jour. Ind. Hygiene, vol. 3, no. 1, pp. 16-28, May 1921.

Hamilton, Alice: Hygienic Control of the Anilin Dye Industry in Europe. U. S. Bur. Labor Statis., Month. Labor Rev., vol. 9, no. 6, pp. 1-21, Dec. 1919.

Hamilton, Alice: Industrial Poisoning in American Anilin Dye Manufacture. U. S. Bur. Labor Statis., Month. Labor Rev., vol. 8, no. 2, pp. 199-215, Feb. 1919.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 93-174.

Hueper, W. C., Briggs, F. A., and Wolfe, H. D.: Experimental Investigation on the Etiology of Aniline Tumors. J. Ind. Hyg. Toxicol., vol. 20, pp. 85-91, 1938.

Hueper, W. C., M. D.: Cancer of the Urinary Baldder in Workers of Chemistry Dye Factories and Dyeing Establishments: A Review. Jour. Ind. Hygiene, vol. 16, no. 5, pp. 255-281, Sept. 1934.

Iszard, Miriam Stewart, M. A.: Determination of Aniline Vapors in the Air. Jour. Ind. Hygiene, vol. 2, no. 7, pp. 259-266, Nov. 1920.

Latishev, L. S.: Professional Diseases of Aniline Industries. Sovetskaya Klin., vol. 15, p. 212, 1931.

McNally, W.: Toxicolory, Industrial Medicine, Publishers, Chicago, 1937, pp. 828-829.

Mollerstrom, J.: Acute Aniline Poisoning. Acta. Med. Scandinav., vol. 71, pp. 73-81, 1929.

Muller, A.: Aniline Tumors in the Region About Basel. Schweiz. Med. Wchnschr., vol. 63, pp. 951-952, 1933.

Newton, C. R.: Industrial Blood Poisons. Jour. Am. Med. Assn., vol. 74, no. 17, pp. 1149-1150, April 24, 1920.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 117-131.

Ohio Department of Health—Aniline and Related Compounds, 1940.

Oppenheimer, Rudolph: Recognition and Treatment of Tumors of the Bladder in Aniline Workers. Zentralbl. f. Gewerbehyg., vol. 8, no. 6, pp. 105-107, June 1920.

Patek, A. J.: Aniline Shoe Dye Poisoning. Jour. Am. Med. Assn., vol. 86, pp. 944-945, March 27, 1926.

Pereyra, G.: Corneal Epithelial Necrosis Due to Anilin Poisoning in the Hat Industry. Rass. di Med. Appl. al Lav. Indust., vol. 6, pp. 134-137, April 1935.

Schutze, W.: Deleterious Action Through the Skin of Poisonous Gases at High Concentrations (Carbon Monoxide, Hydrogen Sulphide, Hydrocyanic Acid, and Aniline). (Arch. f. Hyg., vol. 98, pp. 70-83, 1927.

Schwedtke, G.: Studies on the Formation of Methemoglobin. VIII. The Theory of Methemoglobin Formation from Aniline. Arch. f. exp. Path. u. Pharmacol., vol. 188, pp. 121-129, 1938.

Seglini, C.: Hemoglobin Changes and the Behavior of Porphyrine in Urine and Feces in Experimental Aniline Poisoning. Med. del Lavoro, vol. 29, pp. 289-301, 1938.

Sieben, H.: On Prophylaxis of Tumors of the Bladder Among Aniline and Tar Workers. Med. Klin., vol. 27, pp. 587-588, 1931.

Wade, James L.: Poisoning by Aniline Contained in Shoe Dye. West Va. Med. J., vol. 34, pp. 218-20, 1938.

Williams, J. R., and Challis, F. E.: Methelene Blue as an Antidote for Aniline Dye Poisoning. Jour. Lab. Clin. Med., vol. 19, pp. 166-171, 1933.

Williamson, Raymond, B. Sc., M. B., Ch. B.: The Effect of Aniline Black Dyeing on Factory Workers. Jour. Ind. Hygiene, vol. 4, no. 12, pp. 507-517, April 1923.

Young, A. G.: Haemetological Studies of Aniline Poisoning. Jour. Pharmacol. and Exper. Therap., vol. 27, p. 125, 1926.

Young, A. G., Muehlberger, C. W., and Meek, W. J.: Toxicological Studies of Acute Aniline Poisoning. 1. Experimental Studies of Acute Aniline Poisoning. Jour. Pharmacol and Exper. Therap., vol. 27, p. 101, 1926.

ANTIMONY: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Antimony) Sb, antimony regulus.
(Antimony hydride), SbH_3, stibine, antimoniuretted hydrogen.

A. **Principal Properties:**

1. Antimony.
   a. Gray metal sometimes found native. Sp. gr. 6.5 to 6.86 (variously stated): m.p. 630°C.; b.p. 1440°C. Soluble in acids.
2. Antimony hydride.
   a. Colorless gas. Sp. gr. 2.26 at 25°C.; m.p. –88°C.; b.p. –18°C. Solubility, 0.41 parts in 100 parts of cold water. Wt. per liter, 5.19 gr.
3. Important Compounds:
   a. Antimony.
      a. Antimony-chloride, antimony cinnabar, antimony-crocus, antimony fluorides, antimony-glass, antimony lactate, antimony-ocher, antimony oxides, antimony-oxychloride, antimony oxysulfide, antimony-potassium tartrate, antimony-red, antimony salt, antimony sulfate, antimony sulfide; antimony, sulfurated; antimony tribromide, antimony white, antimony yellow.

B. **Principal Industrial Uses:**

1. Antimony.
   a. Various alloys (type metal, Brittania metal, sterotype metal, bearing metal, pewter): bath tub enamels; antimony compounds.
2. Antimony hydride.
   a. By-product where antimony containing metals are subjected to reducing conditions.

C. **Poisoning—Systemic:**

1. Symptoms.
   a. Antimony.
   b. Symptoms of occupational antimony poisoning are difficult to define. Most of the antimony of industry contains traces of arsenic and it is practically always used in conjunction with lead. It is evident that under such conditions a clear clinical picture would be impossible. However, symptoms of antimony poisoning have been described as follows: tightness of the chest, cough, swelling of the throat, gastro-intestinal disturbances, pustular eruptions especially the scrotum, difficult urination, loss of sexual desire, eosinophilia, and nervous symptoms of many different varieties.
2. Antimony hydride.
   a. The toxic action of antimoniuretted hydrogen or stibine is similar to arsine but less potent. It attacks the central nervous system and the blood. The symptoms of acute poisoning are headache, weakness, nausea, retarded breathing, weak, slow, and sometimes irregular pulse, lowered temperature, and diuresis. Antimoniuretted hydrogen is often encountered as a by-product as in the preparation of hydrogen from zinc. Other toxic impurities such as arsine frequently appear in the same processes.
2. Blood Changes.
   a. Reduction of neutrophils.
   b. Increase in lymphocytes, chiefly small.
   c. Initial decrease in red cells followed by a subsequent increase.

D. **Sanitary Corps Officer:**

1. Control measures.

SELECTED REFERENCES

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 96-98.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 583-584.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 285-290.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 155-159.

Ohio Department of Health, Antimony and Its Compounds, 1940.

ARSENIC: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Arsenic) As_4.
(Arsenic chloride) $AsCl_3$, arsenic trichloride, butter of arsenic, caustic oil of arsenic.
(Arsenic trioxide) As_2O_3, arsenious acid, arsenious oxide, arsenious anhydride, white arsenic.
(Arsine), AsH_3, arsenic trihydrate, arseniuretted hydrogen, arsenious hydride, arsenia, hydrogen arsenide.
(Ethyl arsine) C_2H_5-AsH_2.
(Copper arsenite), $CuHAsO_3$, arsenite of copper, copper orthoarsenite; cupric arsenite, Sheele's green.
(Copper aceto arsenite) $(CuOAs_2O_3)_3$-$Cu(C_2H_3O_2)_2$, acetic arsenite of copper; cupric aceto arsenite; emerald green; emperor green; imperial green; Kaiser green; King's green; meadow green; mitis green; moss green; new green; Paris green; parrot green; patent green; Schweinfurth green; Vienna green.
(Cacodylic acid) $(CH_3)_2AsO$-OH, dimethyl arsenic acid; kakodylic acid.
(Cacodyl) $((CH_3)_2As)_2$, alkarsin.

A. Principal Properties:

1. Important Compounds.
   a. (Arsenic) Arsenous acid; arsenic bisulfide; arsenic, black; arsenic bromide; arsenic chloride; arsenic iodide; arsenic pentasulfide; arsenic pentoxide; arsenic trioxide; arsenopyrite; arsine; arsphenamine; diphenyl chloroarsine.
2. Arsenic.
   a. Silvery brittle crystalline metal turning black in air. Sometimes found native. Sp. Gr. 5.727 at 14°C.; m.p. 814°C. at 36 atm.; subl. 615°C.; hardness 3.5. Insoluble in water; soluble in nitric acid.
3. Arsenic Chloride.
   a. Colorless oily liquid. Sp. Gr. 2.163; m.p. –18°C.; b.p. 130.2°C., wt. per liter of vapor, 7.55 gr.
4. Arsenic Trioxide.
   a. White amorphous, odorless, tasteless powder; poisonous. Sp. Gr. 3.865; m.p. 200°C. Soluble in water, alcohol, acids, and alkalis. Wt. per liter of vapor 8.23 gr.
5. Arsine.
   a. Colorless gas; extremely poisonous. Sp. Gr. 2.695; m.p. –113.5°C.; b.p. –55°C., decomposes at 230°C.; wt. per liter 3.24 gr.
6. Ethyl Arsine.
   a. Colorless liquid. Sp. Gr. 1.217 at 22°C.; b.p. 36°C. Slightly soluble in water.
7. Copper Arsenite.
   a. Fine, light green powder; poisonous. m.p.; decomposes. Soluble in acids; insoluble in water.
8. Copper Aceto Arsenite.
   a. Emerald green powder; poisonous. Soluble in acids; insoluble in alcohol and water.
9. Cacodylic Acid.
   a. Colorless, odorless, deliquescent crystals; poisonous. M.P. 200°C. Soluble in water and alcohol.
10. Cacodyl.
    a. Colorless oil. Sp. Gr. more than 1; m.p. –6°C., b.p. about 170°C. Very slightly soluble in water; soluble in alcohol and ether. Compounds are noted for their vile odor and poisonous properties.

B. Principal Industrial Uses:

1. Arsenic.
   a. Medicine (mercury amalgam); arsenic salts; metallurgy glass.
2. Arsenic Chloride.
   a. Manufacture of carbon tetrachloride from carbon disulfide (catalyst), pharmaceuticals (arsenated albumins, drug), ceramics (luster finishes).
3. Arsenic Trioxide.
   a. Manufacture of pigments, glass, shot and bullets; insecticides, rat poison, cattle dip, weed killer, hide preservative; medicine (violent irritant); manufacture of other arsenic compounds, ceramic enamels, aniline colors, mixed with soda ash for boiler compound, textile mordant, sterilizing agent in water purification.

4. Arsine.
   a. Organic synthesis.
5. Copper Arsenite.
   a. Pigment (paints, wall paper, calico printing); insecticide.
6. Copper Aceto Arsenite.
   a. Pigment, insecticide, wood preservative preparations.
7. Cacodylic.
   a. Synthesis of dyes, drugs and perfumes.

C. **Poisoning—Systemic:**

1. Modes of Entry.
   a. Inhalation.
   b. Ingestion.
2. Symptoms.
   a. Pure Arsenic does not appear to be toxic, but its compounds are exceedingly poisonous. Arsenic containing its oxides should be considered harmful; the sulfide of arsenic, if pure, are insoluble and thus not toxic, but commercial products always contain amounts of arsenic acid, the fatal dose of which varies between 1 and 12 cg. according to Lehmann.

   Industrial poisoning by arsenic is generally chronic and is usually brought about by the inhalation of arsenical dusts. Acute conditions are at times seen due to release of arsine (arseniuretted hydrogen) in industrial processes.

   The clinical signs of arsenic poisoning are generally grouped around the toxic effects on the digestive system, the liver, kidneys, circulatory system and heart. In acute attacks, symptoms of the gastric and renal system predominates; in chronic attacks, symptoms referable to the nervous system and a local action on the skin.

   A very acute form arising from massive ingestion of arsenious acid is described, but it is exceptional in industry. Gastric symptoms are severe; there is abdominal colic, nausea, incessant vomiting, and a feeling of dryness in the mouth and thirst. A few hours later, diarrhea, suppression of urine and anuria supervenes, and death advances rapidly, the patient being pale and cyanosed, the appearance resembling cholera.

   The acute form is more frequent and is seen in cases of suicide, accidents and occasionally industrially. The dose is high and death usually supervenes in a few days to two weeks, but because of early vomiting the poison may be largely rejected and a cure follow. Nausea, vomiting and diarrhea are frequent but usually cease in 24 to 48 hours. Of importance is the fact that the patient then shows a frequent remission of symptoms, improvement generally occurring with a feeling of well-being. However, in a day or two the general conditions again become worse with various skin eruptions (scarlatiniform, morbiliform, urticarial purpuric, etc.), labored breathing, cyanosis, cold extremities, small weak pulse, the urine is loaded with albumen and death follows in a state of syncope. If a cure takes place, convalescence is long with frequent gastro-intestinal trouble, chronic nephritis, and often paralysis; but fatal syncope may occur even after a long period of time.

   The chronic form is the most frequent seen industrially and follows daily doses of the poison insufficient to cause death but producing organic changes. The first signs are digestive troubles, salivation, nausea, vomiting, abdominal colic, diarrhea, pains in the bones, toxic polyneuritis of the small muscles of the hands and feet, signs of paralysis in lower limbs with numbness, tingling or itching, and anesthesia. If the poisoning continues, various skin eruptions occur, palmo-planter keratosis, and bronzing of the skin (melanodorma). The nails show trophic changes and the hair may fall out. Signs of a cold with laryngitis, hoarseness and cough may be present. Cerebral symytoms of a toxic delirium evidenced by a mental confusion, Korsakoff syndrome, etc., may occur. Motor affections are rarely seen industrially; they consist of peripheral paralysis starting in the lower limbs and progressing upward to involve the upper limbs in about one-half of the cases with abolition of reflexes and frequently anesthesia.

   Local action occurs at the point where caustic derivatives of arsenic frequently contact as ulceration of the finger ends, corners of the mouth or genital organs. Painless ulceration of the septum of the nose is frequently due to the arsenious acid which forms when arsenic dust contacts the moist mucous membranes.

Authorities differ as to the role of arsenic in the production of cancer. The researches of certain experts (Bayet, Slosse, etc.) tend to show that arsenic has a cancerigenetic action. This view is not accepted by some German and English authorities. The English view is that arsenic present in tar does not play a role in the production of epitheliomatous ulceration among tar and pitch workers. Delepine showed that arsenic can be found in the hair and urine of persons exposed to toxic vapors. It has been found by chemical analysis in various organs as the liver, intestines, kidneys, brain, etc.

In considering the action of arsenic, account must be taken of such things as intolerance of acclimatization, age, state of digestive tract, amount of fatty food in the diet, etc.

b. Arsenic Trioxide: Historically, it is the most important of the poisons used for criminal purposes. It is a powerful poison and it is stated that two grains may be fatal. It produces inflammation of the stomach and bowels, violent purging and vomiting, profound nervous collapse, hemolysis with jaundice, urine scanty, bloody or suppressed. If death does not occur, there follows sensory nervous disturbances, neuralgic pains, paresthesias; later, paralysis, loss of hair, deformities of the nails, all sorts of skin lesions, and laryngo-bronchial catarrh. Perforation of the nasal septum has been reported in men handling arsenic trioxide.

c. Arsenic Chloride: There may be symptoms of intense irritation, itching or ulceration of the skin and mucous membranes, multiple neuritis with sensory and motor disturbances; sweating of palms and soles, and loss of hair, etc. Delepine showed experimentally that arsenic trichloride applied to the skin could set up poisoning.

d. Copper Arsenite: Symptoms of arsenic poisoning.

e. Copper Aceto Arsenite: Symptoms of arsenic poisoning.

f. Arsine: Arsine may be formed as a by-product in industry when nascent hydrogen present reacts with traces of arsenic in metals or acids, etc. It has an unpleasant garlic smell and is extremely toxic, being a powerful hemolytic poison. It is stated that the early symptoms are those of anoxemia; the later symptoms being largely those due to the effort of the body to excrete the debris of red cells which clog the liver and kidneys. A period of time is required after the inhalation of the gas before the products of hemolysis become evident and symptoms result. This period of time varies with the doses from 6 to 36 hours, according to Legge.

It is stated that an atmosphere of 1 part arsine to 4,000 air is rapidly fatal. According to Zangger, a dilution of 1 to 100,000 is poisonous after an absorption of long duration. Dubitski found arsine to be 10 to 20 times more poisonous than carbon monoxide.

In slight poisoning, there is simply lassitude, headache, malaise, slight dyspnea, occasionally fainting, weak, quick pulse, fall in blood pressure, and sometimes a yellowish coloration of the skin with presence of slight amounts of arsenic in the urine (5 cases have been reported by Wignall). In very slight cases, a cure is effected in a few days after elimination of the exposure.

In average or moderate poisoning, there occurs about 4 to 9 hours after inhalation, lassitude, vertigo, shivering, nausea, vomiting, diarrhea, syncope, oppression and pain in the gastric renal or hepatic regions. Hemoglobinuria appears in 4 to 6 hours; the urine is reddish brown, and for several days contains blood and bile pigments. Jaundice appears in two to three days. Convalescence is fairly long, albuminuria persists, the blood and bile pigments disappear from the urine and strength is slowly regained.

In serious cases, the onset occurs with a feeling of increasing malaise and weakness, shivering, fatigue, headache, nausea, vomiting, paleness, garlic smell on the breath, pain in hepatic and epigastric regions, and kidney tenderness, weakness, somnolence, semi-unconsciousness, giddiness, with restlessness and insomnia and dry throat and thirst. There is anoxemia, cyanosis, dyspnea, increased respiration, and syncope may result. After a few hours (about 8-12), evidences of hemolysis appears, the urine is bloody and contains albumin; it may decrease in quantity, oliguria developing into anuria accompanied by pains in kidney region and convulsions. The pupils may be contracted and react slowly, the conjunctiva may be hemorrhagic or congested. The reflexes may be exaggerated, muscular and nervous pains may occur in the upper or lower limbs. Severe jaundice of the skin and mucous membranes may occur about the second day. On palpatation, the liver, gall bladder and spleen may be noted enlarged and pain elicited in the kidney region. The bile production is increasd several fold. If recovery results, there is slow convalescence with anemia, weakness, and often a toxic polyneuritis.

g. Ethyl Arsine: When high concentrations are inhaled, death results almost immediately. The danger arising from arsenical wall papers is stated to be due to liberation of diethyl arsine from decomposing arsenic in the colors by the action of a mold, penicillum glaucum developing in the starch paste on damp walls.

h. Cacodylic Acid: Action similar to that of ethyl arsine.

i. Cacodyl: Action similar to that of ethyl arsine.

D. Sanitary Corps Officer:

1. Control measures where arsenic or its compounds may become a hazard.

SELECTED REFERENCES

Agasse-Lefont, E., Feil, A., and de Balsac, F. H.: Certain Aspects of Occupational Arsenicism in the Chemical Industry. Chim. et indust., Special no., pp. 945-946, April 1928.

Arsenic. Analysis of Factory Inspection Reports. 1920-1922: III. International Labour Rev., vol. 13, pp. 549-550, April 1926.

Badham, C., Rayner, H. E. G., and Willison, A. M.: Report of Director-General of Public Health, New South Wales for the Year 1934. Division of Industrial Hygiene. Arsenic Poisoning. Section I-E., pp. 116-118.

Berat, A.: Method of Determination of Arsenic Adapted to the Study of Arsenical Products in the Organism. Jour. de pharm. et de chim., vol. 10, pp. 49-69, 1929.

Berlin Letter: The Cause of the Half Disease. Jour. Am. Med. Assn., vol. 84, p. 216, Jan. 17, 1925.

Blondel, R.: Three Cases of Industrial Poisoning by Arseniuretted Hydrogen. Med. du travail., vol. 1, pp. 136-144, 1930.

Case, A. E.: The Estimation of Arsenic in Brewing Materials. Jour. Inst. Brewing, vol. 44, pp. 362-374, 1938.

Cassil, C. C.: Report on (The Determination) Arsenic. Jour. Assoc. Official Agr. Chem., vol. 21, 198-203, 1938.

Dalton, C. H. C.: Arsenical Poisoning. Brit. Med. Jour., vol. 2, p. 297, Aug. 17, 1929.

Delepine, Sheridan, M. B., C. M., M. Sc.: Observations upon the Effects of Exposure to Arsenic Trichloride upon Health. Jour. Ind. Hyg., vol. 14, no. 8, pp. 346-364, Dec. 1922; Jour. Ind. Hyg., vol. 14, no. 9, pp. 410-424, Jan. 1923.

Delephine, Sheridan, M. B., C. M., M. Sc.: Report on Certain Organs in a Case of Fatal Poisoning by Arseniuretted Hydrogen Gas. Jour. Ind. Hyg., vol. 1, no. 7, pp. 356-359, Nov. 1919.

Franke, K. W., and Moxon, A. L.: The Toxicity of Orally Ingested Arsenic, Selenium, Tellurium Vanadium and Molybdenum. Jour. Pharm. Exp. Therap., vol. 61, pp. 89-102, Sept. 1937.

Frohn, W.: Industrial Arsenic Poisoning in Wine Growers. Munch. med. Wochschr., vol. 95, pp. 1630-1635, 1938.

Garmsen, H.: Industrial Arsine Poisoning. Samm. v. Vergiftsf., vol. 8, pp. 67-70, 1937.

Gaudy, F.: The Colorimetric Determination of Arsenic in Toxicology. Its Determination as Colloidal Arsenious Sulfide. Bull. soc. chim. biol., vol. 20, pp. 1102-1107, 1938.

Gaudy, Fernando V. M.: Colorimetric of Arsenic in Toxicology, Rev. brasil. chim. (Sao Paulo), vol. 6, pp. 205-207, 1938.

Gerbis, H.: Three Fatal Cases of Industrial Poisoning with Arseniuretted Hydrogen. Munchen, med. Wchnschr., vol. 72, pp. 1378-1380, Aug. 14, 1925.

Gillert, E.: Arsino Poisoning. Samm. v. Vergift., vol. 7, pp. 89-92, 1936.

The Haff Disease: Lancet, vol. 1, p. 566, March 14, 1925.

Hamilton, A.: Industrial Toxicology, Harper and Brothers, New York, 1934, p. 64-72.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 138, 186-188.

Hopmann, A.: A Fatal Case of Acute Yellow Atrophy of the Liver from Industrial use of Nitrobenzol, Nitrotoluol, or Arsenic? Zentralbl. f. Gewerbehyg., N.S., vol. 4, p. 423, Dec. 1927.

Hopmann, A.: Arsenic in Large Chemical Works. Zentralbl. f. Gewerbehyg., N.S., vol. 3, pp. 126-127, May, 1926.

Lelsall, A., and Campbell, J. H.: The Inhalation of Arsenical Insecticide Dusts. Sci. Agr., vol. 4, pp. 159-161, 1924.

King, W. Bernard, and Brown, F. E.: Modification of Bettendorff's Arsenic Test. II. Catalyzed by Mercury. Jour. Am. Chem. Soc., vol. 31, pp. 968-969, 1939.

Klein, A. K., and Vorhes, F. A. Jr.: Determination of Arsenic (In Foods). Jour. Asssoc. Official Agr. Chem., vol. 22, pp. 121-130, 1939.

Kober, G., and Hayhurst, E.: Industrial Health, P. Blakiston's Son and Co., 1924, p. 303-320.

Korenman, I. M.: Determination of Arsenic. Lab. Prakt. (U. S. R. R.) no. 4, pp. 23-24, 1938.

Kraetzer, A. F.: Raynaud's Disease Associated with Chronic Arsenical Retention. Report of Case Cured by Sodium Thiosulphate. Jour. Am. Med. Assn., vol. 94, pp. 1035-1037, April 5, 1930.

Kremer, D.: An Unusual Case of Arsine Poisoning. Gasmaske., vol. 3, pp. 109-111, 1931, in Chem. Abstr., vol. 26, p. 935, Feb. 20, 1932.

Labse, R.: Poisoning with Arsine. Deutsch. med. Wchnschr., vol. 52, p. 2192 (conclusion), Dec. 24, 1926.

Laborde.: Fatal Poisoning Probably due to Arseniuretted Hydrogen, Soc. de med. legale de Paris, Nov. 14, 1927.

Lawetzky: Epidemic of Muscular Rigidity. Deutsch. med. Wchnschr., vol. 50, p. 1444, Oct. 17, 1924.

Leitch, A., and Kennaway, E. L.: Experimental Production of Cancer Arsenic. Brit. Med. Jour., vol. 2, pp. 1107-1108, Dec. 9, 1922.

Lentz, O.: Haff Disease. Med. Klin., vol. 21, p. 4, Jan. 4, 1925.

Lewin, L.: Haff Disease. Deutsch. med. Wchnschr., vol. 51, p. 133, Jan. 23, 1925.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937. pp. 219-266.

Meyer, E., and Heubner, W.: Observations on Arsine Poisoning. Biochem. Ztschr., vol. 206, pp. 212-222, 1929.

Montgomery, D. W., and Culver, G. D.: Arsenical Pigmentation of the Skin. Med. Rec., vol. 101, pp. 665-667, 1922.

Morris, Herman J., and Wallace, Edward W.: The Storage of Arsenic in Rats Fed a Diet Containing Calcium Arsenate and Arsenic Troxide. Jour. Pharmacol., vol. 64, pp. 411-419, 1938.

Morrison, J. A. S.: Poisonous Gases in the Leather Industry. Leather World, vol. 30, pp. 886-887, 1938.

Naeslund, C.: Measures Against Arsine and Phosphine Poisoning. Upsala Lakaref. Forh., vol. 32, p. 367, May 31, 1927.

Occupation and Health. International Labour Office, Geneva, 1934. Vol. 1, p. 154-176.

Ohio Department of Health, Arsenic and Its Compounds, 1940.

O'Donovan, W. J.: Arsenic Cancer of Occupational Origin. Brit. Jour. Dermat. and Syph., pp. 477-481, Nov. 1924.

Pein, H. v.: Skin Cancer From Arsenic Poisoning in Insect Control. Deutsch. med. Wchnschr., vol. 64, pp. 565-568, 1938.

Rosenow, G., and Tietz, L.: Haff Disease. Klin. Wchnschr., vol. 3, p. 1991, Oct. 28, 1924.

Seeger, F., and Tidow, G.: Haff Disease. Munchen, med. Wchnschr., vol. 71, p. 1455, Oct. 17, 1924.

BENZENE (BENZOL): TOXICITY AND POTENTIAL DANGERS

A. Principal Properties — Physical-Chemical:

1. Colorless liquid which solidfies at 5.5° C. and boils at 79.7° C.
2. Soluble in water to the extent of 0.06 parts per hundred at 20° C. and mixes freely with alcohol and ether.
3. Commercial benzene is seldom pure and may be contaminated with xylene, toluene, phenol, thiophene, carbon disulphide, acetonitrile, pyridine and other substances.
4. Chemically pure Benzene has a flash point of −12° to +10° C. Ignition may be caused by open flames or sparks from electrical appliances.

B. Principal Uses:

1. Chemical Industry. Prepared by the distillation of coal and coal tar and it is used for the extraction of oils and fats, in the synthesis of organic chemicals, such as dyes and their intermediates, in the manufacturing of varnishes, lacquers, stains and paints.
2. In the Rubber Industry and in many other industries it is used in certain rubber cements.
3. In the dry-cleaning industry it played an important role prior to the introduction of chlorinated hydrocarbons for that purpose.

C. Poisoning—Systemic:

1. Mode of entry.
   a. Mainly absorbed by the lungs.
   b. Accidental ingestion.
2. Excretion.
   a. Oxidized in the organism with the formation of phenol and diphenols, which in turn are conjugated with sulfuric acid and excreted in the urine, thus reducing the amount of inorganic sulfates.
3. Acute Poisoning.
   a. Inhalations of large quantities of Benzene causes inebriation, soon followed by fatigue, sleepiness, ringing in the ears, vertigo, nausea, vomiting, headache and staggering gait.
   b. Prolonged inhalation will cause twitching, tonic and clonic convulsions, paralysis and loss of consciousness may result. With very large doses, unconsciousness, convulsions and death due to respiratory paralysis may occur very rapidly. Depending upon the duration of the unconsciousness and the severity of the circulatory failure, nervous disturbances of different nature may be observed as aftereffects of such poisoning.

D. Chronic Poisoning:

1. The continued exposure to small quantities of Benzene may cause such subjective symptoms as fatigue, somnolence, headache, vertigo, general debility, pupillary abnormalities and gastro-intestinal disturbances.
2. In the early stages of the Benzene poisoning, hemorrhages under the skin and the mucous membranes may occur. Bleeding from the gastro-intestinal tract and from the uterus may be observed. The urine may contain albumin, casts, and bile pigments, indicating injury of the kidneys and liver.
3. The contact of Benzene with the skin may cause erythema, dry scaling, and occasionally, vesicular papules. Benzene is a fat solvent. It removes the protective fat of the skin and predisposes to secondary infections. More prolonged exposure may result in injury resembling first and second-degree burns.

E. Laboratory Findings:

1. W.B.C.—5,000 or 5,500 or less with a diminution of polynuclears is usually considered as incipient benzene poisoning.
2. Secondary anemia usually occurs after toxic effects on the white blood cells have become manifest and it is associated with corresponding change of the hemoglobin content.
3. The ratio of inorganic to total sulfates should be determined, a reduction of which will indicate the existence of exposure to benzene. In case, upon repeated examinations, the percentage of organic sulfates is 30% or more, the concentration of benzene in the air of such operation should be determined and reduced by proper engineering methods.

F. Toxic Concentrations:

1. The maximal allowable concentration of Benzene (Benzol) is at present accepted as 100 parts per million by volume of air (corresponding to 0.32 milligram per liter at 25° C. and 760 mm. Hg) for exposures not exceeding a total of eight (8) hours daily.

G. Pathology:

The most characteristic pathological changes are seen in the bone marrow, which shows a variety of pictures varying from hyperplasia to hypoplasia, and may result in complete aplasia of the myeloic cells. This effect may not be solely restricted to the bone marrow but may extend also to the germinating structures of the lymphatic glands, the pulp of the spleen, the Peyer's plaques, and the cortex of the thymus. Degenerative changes may occur in the liver, kidneys and heart, varying in intensity with the concentration and the duration of the exposure. The prognosis becomes bad when the leucocytes drop to or below 1,000. There may be a moderate anisocytosis. The formation of hemorrhages may be partly explained by an increase of the clotting time, perhaps on account of a reduction of the thrombocytes. It is partly due to vascular damage; the walls of the blood vessels may show degenerative changes of variable intensity, from turbid swelling to fatty degeneration, as may be seen in other organs. Unlike most other poisons, continued exposure to benzene does not increase the resistance of the organism towards its toxic effects. On the contrary, repeated subtoxic exposure may finally result in sudden and severe injury, as has been observed repeatedly.

H. Sanitary Corps Officers:

1. Frequent checks of local exhaust systems.
2. Determination of Benzene (Benzol) in air should be taken at sufficient intervals of time so that any variations of concentrations will be evident, and in sufficient number to avoid any reasonable doubt of the results found.

I. Medical Control:

1. Preemployment
   a. Juvenile workers appear more susceptible than adult males.
   b. Chlorosis, tuberculosis and pregnancy; also organic heart disease, tendency to hemorrhages and anemia, should be excluded from this type of hazard.
2. Periodic examination every six (6) months with especial attention to the total white cell count, differential count and observing the relation of neutrophil to lymphocytes. Decrease of leucocytes below 5,000 with relative lymphocytosis or eosinophilia should be a contra-indication to further exposure. Urinary determination of urinary sulphates when percentage of organic sulphate is 30% or more indicates exposure to Benzene. The urinary test should be made every month.

J. Instructions to Operators:

1. Cleanliness of the operation.
2. Adequate provisions for protection of workers in case of spillage.
3. When in enclosures, no person should enter without proper safety appliances, such as air-supplied masks and safety belts.

K. Suggested Benzol (Benzene) Periodic Check:

1. Name, Address, Department, Age, Race, Sex and Duration of Exposure.

2. Acute Symptoms and Dates.
   - *a.* Inebriation
   - *b.* Fatigue
   - *c.* Sleepiness
   - *d.* Tinnitus
   - *e.* Vertigo
   - *f.* Vomiting
   - *g.* Headache
   - *h.* Staggering Gait
   - *i.* Twitching
   - *j.* Tonic Convulsions
   - *k.* Clonic Convulsions

3. Chronic Poisoning.
   - *a.* Fatigue
   - *b.* Somnolence
   - *c.* Headache
   - *d.* Vertigo
   - *e.* General Debility
   - *f.* Gastro-intestinal Disturbances

4. Laboratory Findings.
   - *a.* Total White Blood Count
   - *b.* Differential
   - *c.* Total Red Blood Count
   - *d.* Hemoglobin

5. Urine—Organic Sulphates.

6. Remarks.

SELECTED REFERENCES

Journal American Medical Association, 12 September 1936, Benzine Exposure.

Treasury Department, U. S. P. H. S., 1926, Benzol Poisoning as an Industrial Hazard.

National Institute of Health, Division of Industrial Hygiene, 14 March 1941. Benzene (Benzol), Its Toxicity and Potential Dangers.

BENZENE: ALKYL DERIVATIVES

Synonyms:

(Toluol)—Toluene, methylbenzene, methylbenzol, phenylmethane.
(Ethyl Benzol)—Phenylethane, ethylbenzene.
(Xylol)—Xylene.

Uses:

Toluol:—Organic preparations: benzoic acid, dyes, perfumes, toluidines, tolidines, saccharin, explosives (TNT).

Ethyl Benzol:—Organic synthesis, anti-knock for motor fuel, lacquer diluent, spotting cellulose acetate silks.

Xylol:—Organic preparations; solvent, meta-xylidine, microscopy, artificial musk, removal of naphthalene from illuminating gas; preventing stopping up of gas pipes with naphthalene; lacquers and varnishes (solvent); solvent for rubber cements.

Mode of Entry:

Toluol, Ethyl Benzol and Xylol:—Inhalation and ingestion.

Symptoms:

Toluol, Ethyl Benzol and Xylol:—The toxic action is considered similar to benzol, but has a stronger irritant action on mucous membranes. There may be symptoms as vertigo, exaggerated reflexes, confusion, hallucination and psychogenic disturbances.

Toxic Concentrations:

(Toluol—Toluene) The maximal permissible is 200 parts per million for an eight (8) hour period, although this concentration may produce slight muscular coordination, thus reducing the individual more prone to accidents.

SELECTED REFERENCES

Batchelor, J. J.: The Relative Toxicity of Benzol and Its Higher Homologues. Am. Jour. Hyg., vol. 7, pp. 276-298, May 1927.

Bormann, G.: Diagnosis and Therapy of Chronic Benzene Poisoning. Arch. f. Gewerbepathe. u. Gewerbehyg., vol. 8, pp. 194-205, 1937.

Engelhardt, W. E., and Estler, W.: Acute Narcotic Action of Aliphatic and Aromatic Hydrocarbons. I. The Action of Single Inhalations of Different Concentrations of Benzene, Benzol, Toluol, and Xylol on Rabbits and Cats. Estler, W.: II. Repeated Inhalations of Benzene, Benzol, Toluol, and Xylol in White Mice. Arch. f. Hyg., vol. 114, pp. 249-260, and pp. 261-271, Aug. 1935.

Fuhner., and Pietrusky, F.: Butylacetate: Chronic Occupational Toluol Poisoning: Sequelae? Samm. von Vergiftungsfallen., vol. 5, Lieferung 1, 1934.

Gorkin, S. D., and Kagan, E. M.: The Influence of the Nitro and Amido Derivatives of Benzene On the Organism. Experimental and Clinical Research. Ukrainian Inst. of Path. and Hyg. of Work., vol. 14, Paper. p. 318, Ukrainian State Med. Publ. Co., Karkov, 1935.

Hamilton, A.: Industrial Toxicology. Harper and Bros. Publishers, New York, 1934, pp. 165-167.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 158-165.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., Publishers, Philadelphia, 1924, pp. 508-509.

Luszczak, A., and Hammer, E.: Toluol, benzol, thymol: spectrographic determination. Jour. Ind. Hyg., vol. 15, pp. 162-163, 1933.

Occupations and Health. International Labour Office, Geneva, 1934, pp. 1070-1073, 1276.

Ohio Department of Health, Alkyl Derivatives of Benzol, 1940.

CADMIUM

A. Principal Properties:

1. Cadmium is ductile, malleable and capable of taking a high polish.
2. Atomic weight 112.41 and a density of 8.6 gm. per cc. at 20° C.
3. It is a silvery-white metal with a bluish tinge.
4. When heated in air, cadmium volatilizes and burns with a bright flame emitting an abundance of brown oxide (CdO) fume.

B. Principal Industrial Exposures:

1. In electro-plating operations.
2. Smelting of cadmium ores.
3. Welding of alloys.
4. Spraying of cadmium-bearing paints and pigments, manufacture of cadmium compounds, melting the metal, and cadmium processes particularly of marine hardware and other fittings which were formerly zinc coated.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation of fumes, vapor or dust.
   b. Ingestion from food products that have been produced or processed in cadmium-plated vessels or molds. The metallic-cadmium coating on utensils used for food dissolves when in contact with a weak acid solution.
2. Symptoms
   a. Acute by inhalation
      (1) Dryness of throat, cough, headache, vomiting and a sense of constriction of the chest.
      (2) Later symptoms are predominantly referable to the respiratory system and are characterized by cough, pain in the chest, severe dyspnea, and prostration. These symptoms result from a pneumonitis which in many instances is followed by broncho-pneumonia. A few have symptoms referable to the gastro-intestinal system.
   b. Acute by ingestion
      (1) Increased salivation.
      (2) Choking attacks.
      (3) Persistent vomiting.
      (4) Abdominal pain.
      (5) Diarrhea and tenesmus.

D. Pathology:

1. Post mortem examinations revealed edema, congestion, hemorrhage and partial collapse of the lungs. Cloudy swelling of cells of the liver and kidneys were noted. In one case, there was also congestion of the spleen, fatty infiltration of the pancreas, and slight chronic gastritis.

E. Toxic Concentrations:

1. The maximum allowable concentration of Cadmium or of its compounds in air has been accepted as 1 milligram of cadmium per 10 cubic meters of air.

F. Sanitary Corps Officers:

1. Prevention of industrial cadmium exposure depends upon the type of process involved in which cadmium fumes are generated. Proper exhaust ventilation or the use of positive pressure masks is recommended, the latter especially where the cadmium content is high.

Alsberg, C. L., and Schwartze, E. W.: Pharmacological Action of Cadmium. Jour. Pharmacol. and Exper. Therap., Proc., 13:504, 1919.

Bulmer, F. M. R., Rothwell, H. E., and Frankish, E.: Industrial Cadmium Poisoning. A Report of Fifteen Cases Including Two Deaths. Canadian Pub. Health J., 29:19, 1938, Abst. Jour. Ind. Hyg., 20:123, June, 1938.

Dublin, L. I., and Vane, R. J.: Occupation Hazards and Diagnostic Signs. U. S. Department of Labor, Bureau of Labor Statistics, Bulletin, 582; 32, September, 1933.

Fairhall, L. T., and Prodan, L.: The Colorimetric Determination of Minute Amounts of Cadmium. Jour. Am. Chem. Soc., 53:1321, 1931.

Ficklen, J. B.: Manual of Industrial Health Hazards. Service to industry, Publishers, West Hartford, Conn., 1941, pp. 57-59.

Flury, F., and Zangger, H.: Lehrbuch der Toxikologie fur Studium und Praxis. Berlin, Julius Springer, 1928.

Hamilton, A.: Industrial Toxicology, Harper & Bros. Publisher, New York, 1934, pp. 104-06.

Industrial Cadmium Poisoning. Ohio Industrial Hygiene Bulletin, Ohio Department of Health, Columbus, Ohio, 1, 2, May, 1939.

Katz, S. H., Smith, G. W., Myers, W. M., Trostell, L. J., Ingels, M., and Greenburg, L.: Comparative Test of Instruments for Determining Atmospheric Dusts. U. S. Pub. Health Bull. 144, 1925.

Legge, T. M.: Cadmium Poisoning. Ann. Rep. Chief Inspect. Factories for 1923, H. M. Stationery Office 1924, p. 74.

Lehmann, K. B.: Kurzes Lehrbuch der Arbeits-und Gewerbehygiene. Leipzig, S. Hirzel. 1919, p. 136.

National Institute of Health, Division of Industrial Hygiene.

Occupation and Health. International Labour Office, Geneva (1930) pp. 330-331.

Ohio Department of Health, Cadmium and its Compounds, 1940.

Prodan, L.: Cadmium Poisoning: I. The History of Cadmium Poisoning and Uses of Cadmium, J. Ind. Hyg., 14:132, 1932, II. Experimental Cadmium Poisoning, p. 174.

Schwartze, E. W., and Alsberg, C. L.: Studies on the Pharmacology of Cadmium and Zinc with Particular Reference to Emesis. Jour. Pharmacol. and Exper. Therap, 21:1, 1923.

Schwarz, L.: Kleine Mitteilungen: Gewerbliche Cadmium Vergiftung. Ztschr. f. Gewerbe-Hyg., 31: 190, 1930.

Schwarz, L., and Otto, A. Ist Cadmium ein Gewerbliches Gift? Ztschr. F. Hyg. u. Infektionskrankh., 104: 346, 1925.

Stephens, C. A.: Cadmium Poisoning. J. Ind. Hyg., 2:129, 1920-21.

Tracinski: Die Oberschlesische Zink-industrie and ihr Einfluss auf die Gesundheit der Arbeiter. Deutsch. Vrtljschr. f. offentl. Gsndhtsplg., 20:59, 1888.

CARBON DISULFIDE

Chemical Formula and Synonyms:

(CS_2, Carbon bisulfide)

A. Principal Properties:

1. Clear, colorless, inflammable liquid; almost odorless when pure the commercial article has a strong disagreeable odor; poisonous; Sp. gr. 1.2927; m. p. —111° C., b. p. 46.25° C.; flash point (observed) —25.5° to —20° C. Soluble in alcohol and ether; slightly soluble in water.

B. Principal Industrial Uses:

1. Solvent varnishes, matches, preservative, insecticide, manufacture of carbon tetrachloride, dry cleaning, rubber solvent, viscose rayon manufacture, and chemical synthesis.

C. Systemic Poisoning:

1. Modes of Entry
   - *a.* Inhalation.
   - *b.* Contact with the skin.
2. Symptoms
   - *a.* Acute and chronic types are recognized. Symptoms of acute poisoning are characterized by a stage of well-being, excitement like alcoholic intoxication, hallucinations, sometimes uncontrollable laughter, headache, throbbing of temples, palpitation, fainting and drowsiness, irritability, and insomnia. Digestive disturbances follow the above symptoms; there is nausea, vomiting, loss of appetite, occasionally diarrhea, sometimes colic and constipation. Carbon disulfide dissolves the lipoids of the blood and acts upon the central nervous system and parenchmatous organs. There is weakness of legs, unsteady gait, incoordination, signs of fatigue, loss of memory and a mania with homicidal and suicidal tendencies reported.

Chronic symptoms may appear in a few weeks or months or after years of work. Following the exciting stage there may be a stage of depression, melancholia, faintness, giddiness, drowsiness, exhaustion, headache, drunken gait, and exaggerated reflexes. Usually a positive Romberg is found. Peripheral neuritis, disturbances of digestion, taste, smell, and sight are present. Progressive emaciation, atrophy of muscles, circulatory and respiratory troubles may continue. The patient cannot read because of disturbance of vision, color fields and retrobulbar neuritis.

Recovery may be rapid or it may be slow and extend over several months. Genital disturbances may include; in male, genital hyperaethesia and exaggerated activity followed by impotence; in female, menstrual disturbances, ovarian pain, abortions and breast atrophy. Locally it has an irritant action; contact with the skin causes a sensation of burning and subsequently anaesthesia, later dryness, erythema, eczema or pigmentation. Various cutaneous trophic changes have been described. Sweating may be abundant with the odor of carbon disulfide.

b. Elimination: Mainly through the pulmonary tract and in the urine, also through the sweat and intestinal tract. Elimination is slow, thus a cumulative action may be apparent.

TOXIC CONCENTRATIONS

Acute and Chronic Poisoning

Milligrams per Liter	*Parts per Million*	*Length of Exposure*	*Comment*
0.003	1.0	Work period	Maximum allowable concentration[1, 2]
0.01	3.2	Work period	Maximum allowable concentration[3]
0.06	20.0	Work period	Maximum allowable concentration[4]

[1] Industrial Commission, State of Wisconsin (U. S. A.).
[2] Industrial Accident Commission, State of California (U. S. A.) (suggested).
[3] Union of Soviet Socialistic Republics.
[4] Department of Labor and Industries, State of Massachusetts (U. S. A.) (suggested).

D. Sanitary Corps Officer:

1. Proper control and safe handling.

SELECTED REFERENCES

Amblyopia and the Artificial Silk Industry: Brit. Med. Jour., June 1, 1929, vol. 1, p. 1008-1009.

Baumann, C.: Carbon Disulfide Poisoning of the Nervous System. Ztschr. f. d. ges, Neurol. u. Paychiate., vol. 166, pp. 568-580 (1939).

Blain, Jules: Hygienic Precautions to be Observed in the Manufacture and Industrial Use of Carbon Disulfide. Industrie Chimique, 1920, 7, pp. 311-313.

Castiglioni, A.: Detection of Carbon Disulfide. Z. anal. Chem., vol. 115, pp. 257-259 (1939).

Castiglioni, A.: Estimation of Carbon Disulfide. Ann. Chim. Applicata, vol. 29, pp. 196-198 (1939).

Effects of Alcohol on Workers with Carbon Disulfide, J. A. M. A. (Queries and Minor Notes), vol. 109, 1472 (Oct. 30, 1937).

Ficklen, Joseph B.: Manual of Industrial Health Hazards. Service to Industry, 1940, pp. 62-65.

Flury, F., and Zernik, F.: Schadliche Gase. J. Sprigner, Berlin, 1931, p. 300.

Hamilton, A.: Industrial Toxicology. Harper and Bros. Publishers, New York, 1934, pp. 241-244.

Harrower, J. Roberts, and Wiley, Frank H.: The Determination of Carbon Disulfide in Blood. J. Ind. Hygiene, vol. 19, p. 486 (1937).

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, p. 169.

Kober and Hayhurst: Industrial Health, P. Blakiston's Son & Co., Philadelphia, 1924, p. 648.

Matuszak, M. P.: Iodometric Determination of Carbon Disulfide, Ind. Eng. Chem., Anal. Ed., 4, p. 98 (1932).

McDonald, R.: Carbon Disulfide Poisoning. Arch. Ophthal., vol. 20, pp. 838-845 (1938).

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 738-740.

Occupation and Health, International Labour Office, Geneva (1930), pp. 352-362.

Ohio Department of Health, Carbon Disulfide, 1940.

Survey of Carbon Disulfide and Hydrogen Sulfide Hazards in the Viscose Rayon Industry. Pennsylvania Department, Labor and Industry, Occupational Disease Prevention Division, Bull. No. 46, p. 69, Harrisburg, Pa. (1938).

Weise, W.: Magen-Darm-Erkrankungen durch. Chronische Schwefelkohlenstoff and Schwefelwassertstoff-Inhalation. Arch. Gewerbepath. Gewerbehyg. 4, p. 219 (1933).

CARBON MONOXIDE

A. Principal Properties:

1. Carbon Monoxide, CO, has a molecular weight of 28.01, a specific gravity of 0.9671 (air = 1), and its density is 1.2504 grams per liter (at 0° C. and 760 mm. Hg.). It is a colorless and odorless gas, except in high concentrations when it has an appreciable garlic-like odor. It may be absorbed by dusts, as for instance by coal dust, and may be again liberated under certain conditions. At 650° C. it burns with a blue flame which is extinguished in air containing less than 13.4 percent oxygen, but it does not support combustion. Its explosive limits are between 12.5 and 74.2 volumes percent carbon monoxide in air.

B. Principal Industrial Uses:

1. The sources of exposure are in chemical, metal, garment, ceramic, mining and electric industries; also in those industries which use ovens and stoves, and in homes carbon monoxide poisoning has been reported from gas heaters, etc.

C. Poisoning—Systemic:

1. Modes of entry
   a. Exclusively through the lungs by absorption. Elimination of carbon monoxide takes place solely through the lungs by reversal of the process responsible for its absorption.

2. Symptoms
   a. Acute—From the central nervous system—feeling of fear, headache, vertigo, vomiting, abdominal pain, cough, and later asphyxial convulsions. Freqeuently there is a rise of body temperature which may last several days. The most characteristic sign of carbon monoxide poisoning is the loss of consciousness which may occur quite suddenly and which is usually very persistent so that it takes a comparably long time to recover even under treatment.

   b. Signs of motor irritation are: Spasms, especially of the upper extremities, trismus, choreatic movements, and occasionally convulsions.

   c. Signs of motor depression have been observed in the form of weakness, especially of the legs. This may sometimes persist after the patient has recovered otherwise, and occasionally, may result in paralysis or paresis. Sensory disturbances may consist in headache, pain in the extremities, and in the cardiac region, anesthesias of parts of the body, and neuritides.

   d. Psychic symptoms following acute carbon monoxide poisoning may vary in type and intensity. Frequently there is more or less complete amnesia regarding the time of exposure. Psychic changes, such as restlessness, irritability, sometimes followed by depression, delusions, disorientation, and occasionally, dementia, may persist for some time after other symptoms have subsided.

   e. From the circulatory apparatus:—Lowering of the blood pressure due to vasodilatation and weakening of the heart muscle, or increase of the blood pressure, stasis in the circulatory system, quickening or slowing of the pulse rate and cardiac distress may be observed.

   f. The color of the skin and especially of the mucous membranes is frequently, but not always, bright red and there may be small hemorrhages, localized edema, vesicles with serous content, and a tendency for decubitus and gangrene.

   g. From the gastrointestinal and urinary tract, nausea, vomiting, diarrhea or constipation, incontinence of urine, and bladder spasms have been reported.

   h. Chronic carbon monoxide poisoning:—There is at present considerable controversy as to whether or not there is chronic carbon monoxide poisoning, largely depending on the interpretation of the word "chronic." It appears that continued exposure to moderately toxic concentrations will result in disturbances of the circulation and nervous system.

   i. Symptoms caused by gradual increase of percentages of carbon monoxide hemoglobin in the blood while the individual is at rest or doing moderate exercise.

Blood saturation in percent of CO hemoglobin	*Symptoms*
10-20	Tightness across forehead, possibly slight headache, dilatation of cutaneous blood vessels.
20-30	Headache, throbbing in temples.
30-40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting, collapse.
40-50	Same as previous item with more possibility of collapse and syncope, increased respiration and pulse.
50-60	Syncope, increased respiration and pulse, coma with intermittent convulsions, Cheyne-Stokes respiration.
60-70	Coma with intermittent convulsions, depressed heart action and respiration, possibly death.
70-80	Weak pulse and slow respiration, respiratory failure, and death.

3. Laboratory Procedures
   a. The blood picture shows, occasionally, in the beginning, a more or less high white blood cell count with a relative increase of the polynuclears, and insubacute poisoning, an increase of the red blood cells and hemoglobin.
4. Pathology
   a. The mechanism of carbon monoxide poisoning, most investigators agree, are due to its great affinity for hemoglobin and that it acts mainly by interfering with and finally inhibiting completely the oxygen metabolism.
5. Treatment
   a. According to procedure outlined by R. R. Sayers, as follows:
   b. The victim should be removed to fresh air as soon as possible.
   c. If breathing has stopped, is weak and intermittent, or present in but occasional gasps, artificial respiration by the Shaefer method should be given persistently until normal breathing is resumed or until after the heart has stopped.
   d. Pure oxygen or a mixture of 5 percent carbon dioxide and 95 percent oxygen should be administered using an inhaler, beginning as soon as possible and continuing for at least 20 minutes in mild cases and as long as 3 hours if necessary in severe cases if the patient does not regain consciousness. The administration of oxygen or of the mixture of carbon dioxide and oxygen when given immediately will greatly lessen the number and severity of the symptoms from carbon monoxide poisoning and will decrease the possibility of serious after effects.
   e. Circulation should be aided by rubbing the extremities of the patient and keeping the body warm with blankets, hot-water bottles, hot bricks, or other devices, care being taken that these objects have been wrapped or do not come in contact with the body and cause burns.
   f. The patient should be kept at rest, lying down to avoid any strain on the heart. Later he should be treated as a convalescent and should be given plenty of time to rest and recuperate. Exercise was at one time recommended; however, the procedure is hazardous, as the patient quite often loses consciousness, and in some cases death occurs.
6. Toxic Concentrations
   a. The maximum allowable is 100 parts per million.

D. **Sanitary Corps Officer:**

1. Prevention of pollution of air with carbon monoxide from all areas where there is a known or suspected source. Control measures and determinations of carbon monoxide in air.

SELECTED REFERENCES

National Institute of Health, Division of Industrial Hygiene Carbon Monoxide: Its Toxicity and Potential Dangers, March 7, 1941.

CHLORINATED HYDROCARBONS

Chemical Formula and Synonyms:

(Methyl chloride) CH_3Cl; chloromethane; monochloromethane.
(Methylene chloride) CH_2Cl_2; carrene; dichloromethane; methylene bichloride; methylene dichloride.
(Chloroform) $CHCl_3$; formyl trichloride; methylene trichloride; trichloromethane.
(Ethyl chloride) CH_3CH_2Cl; chloroethane; hydrochloric ether; muriatic ether.
(Ethylene dichloride) $ClCH_2$-CH_2Cl; dichloroethane; dutch liquid; elayl chloride; ethylene chloride.
(Trichloroethane) CH_2Cl-$CHCl_2$; chloroethylene chloride; ethylene chlorochloride; monochlorinated dutch liquid; vinyl trichloride.
(Tetrachloroethane) $CHCl_2$-$CHCl_2$; acetylene tetrachloride.
(Pentachloroethane) $CHCl_2$-CCl_3.
(Dichloroethylene) $CHCl{=}CHCl$.
(Trichloroethylene) $ClCH{=}CCl_2$.
(Tetrachloroethylene) $Cl_2C{=}CCl_2$; carbon bichloride; carbon dichloride; tetrachloroethene.
(Vinyl chloride) $CH_2{=}CHCl$.
(Propylene dichloride) CH_3-$CHCl$-CH_2Cl; dichloroisopropane; propylene chloride.
(Chloroprene) $CH_2{=}CCl$-$CH{=}CH_2$; chlorobuta —1, 3—diene (2).

A. Principal Properties:

1. Methyl chloride
   a. Colorless, non-corrosive, liquifiable gas which is transparent in both the gaseous and the liquid state. Faintly sweet, ethereal odor; non-irritant to eyes or lungs. Sp. Gr. (liquid) 0.998 at —24° C.; sp. gr. (gas) 1.7438.
   b. Soluble in water, alcohol, chloroform.
2. Methylene chloride
   a. Colorless, volatile liquid; poisonous when inhaled. Sp. Gr. 1.336 at 20°/4° C.
   b. Soluble in alcohol and ether; slightly soluble in water. Wt. per liter of gas, 3.53 grams.
3. Chloroform
   a. Clear, colorless, highly refractive, volatile liquid; characteristic odor: non-inflammable. Sp. gr. 1.526 at 0° C.
   b. Miscible with alcohol, ether, benzol, benzine, fixed and volatile oils; slightly soluble in water. Wt. per liter of vapor, 4.97 grams.
4. Ethyl Chloride
   a. Gas at ordinary temperature; compressed, a colorless, highly inflammable, volatile liquid. Sp. Gr. (liquid) 0.9214.
   b. Soluble in alcohol and ether; slightly soluble in water. Wt. per liter of vapor, 2.68 grams.
5. Ethylene Dichloride
   a. Colorless, oily liquid; pleasant chloroform-like odor; sweet taste. Sp. gr. 1.2569 at 20°/4° C.
   b. Soluble in the usual organic solvents; very slightly soluble in water. Wt. per liter of vapor, 4.11 grams.
6. Trichloroethane
   a. Liquid. Sp. Gr. 1.441 at 25.5/4° C.
   b. Insoluble in water; very soluble in alcohol and ether. Wt. per liter of vapor, 5.55 grams.
7. Tetrachloroethane
   a. Colorless liquid. Sp. Gr. 1.600 at 20/4° C.
   b. Soluble in alcohol and ether; insoluble in water. Wt. per liter of vapor, 6.98 grams.
8. Pentachloroethane
   a. Water-white liquid. Sp. Gr. 1.671 at 25/4° C. Wt. per liter of vapor, 8.42 grams.
9. Dichloroethylene
   a. Non-combustible colorless liquid. Sp. Gr. 1.28; wt. per liter of vapor, 4.03 grams.
10. Trichloroethylene
    a. Colorless liquid with a mild characteristic odor; Sp. Gr. 1.472 at 15° C.
    b. Miscible with all common organic solvents. Wt. per liter of vapor, 5.47 grams.

11. Tetracholoroethylene
    a. Colorless liquid; ether-like odor. Sp. Gr. 1.6080.
    b. Soluble in alcohol and ether, insoluble in water. Wt. per liter of vapor, 6.89 grams.
12. Vinyl Chloride (Gas)
    a. Soluble in alcohol. Readily polymerizes in presence of light. Polymerized mass soluble in mesityl oxide.
13. Propylene Dichloride
    a. Colorless liquid; chloroform-like odor. Sp. Gr. 1.166 at 14° C.
    b. Soluble in alcohol and ether; insoluble in water.
14. Chloroprene
    a. Colorless liquid. Sp. Gr. 0.958 at 20/20° C.
    b. Slightly soluble in water; soluble in all proportions in alcohol and ether.

B. Principal Industrial Uses:

1. Methyl chloride:—Refrigerant; medicine; methylating agent.
2. Methylene Chloride:—Fat and resin solvent; paint remover.
3. Chloroform:—Solvent; anesthetic; antiseptic.
4. Ethyl Chloride:—Medicine and dentistry (local anesthetic); organic synthesis; refrigeration; analytical reagent; solvent for phosphorus, sulfur, fats, oils, resins, and waxes; insecticides; used for making lead tetraethyl.
5. Ethylene Dichloride:—Solvent for oils, rubber, cellulose acetate, soaps; degreasing agent; cleaning fluid; fumigant; organic synthesis.
6. Trichloroethane:—Organic synthesis; solvent; anesthetic.
7. Tetrachloroethane:—Solvent; cleansing and degreasing metals; paint removers, varnishes, lacquers, photographic film; resins and waxes; extraction of oils and fats; ethyl alcohol denaturant; organic synthesis.
8. Pentachloroethane:—Solvent; organic synthesis.
9. Dichloroethylene:—Solvents; anesthetic; antiseptic; organic synthesis; fermentation retarding agent; analytic reagent; refrigerant.
10. Trichloroethylene:—Fat extraction; perfumes; textiles; foods, paints and varnish; leather, glues and gelatins; soap; dry cleaning; rubber; extraction of miscellaneous organic substances and manufacture of various organic chemicals; degreasing leather and metals.
11. Tetrachloroethylene:—Organic preparations; solvent; pharmaceuticals; dry cleaning soaps; detersive compounds.
12. Vinyl Chloride:—Manufacture of plastics, varnishes, electrical insulating material.
13. Propylene Dichloride:—Solvent for oils, fats, waxes, rubber and gum; synthesis; cleaning solution.
14. Chloroprene:—For Artificial Rubber.

C. Poisoning—Systemic:

1. Modes of Entry.
    a. Industrial poisoning by the chlorinated hydrocarbons is concerned chiefly with the inhalation of their vapors. Occasionally, absorption through the skin may be encountered, but ingestion is rarely indicated as a means of industrial poisoning.
2. Symptoms.
    a. Methyl Chloride:—Methyl Chloride is a reactive gas and in the blood is slowly converted to methyl alcohol and sodium chloride. Consequently methyl alcohol continues to accumulate in the body as long as the exposure to methyl chloride persists. Concentrations too small to exhibit the anesthetic action of undecomposed methyl chloride may produce symptoms of poisoning similar to those of methyl alcohol. A series of case studies on workers exposed to methyl chloride in the refrigeration industry revealed symptoms which are listed in order of frequency encountered as follows: Attacks of vertigo, drunken gait, feeling of lightness, somnolence, ptosis of the pupils, anorexia, nausea, loss of weight, eye trouble including diplopia. As these symptoms diminished, persistent insomnia set in with trembling of the extremities in several cases. Formates were found in the urine, the amount usually corresponding with the severity of the poisoning. The evolution of the poisoning was slow and the recovery delayed. The introduction of new and less toxic refrigerants has curtailed the use of methyl chloride in the refrigeration industry.

b. Methylene Chloride:—Methylene Chloride is an anesthetic with properties similar to those of chloroform. Its use as a paint remover in unventilated rooms has given rise to symptoms of mild anesthesia. The victims experienced severe pains and tingling in the legs and arms, headache, vertigo, stupidity, drowsiness, anorexia, precordial pain, and fatigue on exertion.

c. Chloroform:—The anesthetic properties of chloroform are well known to all physicians. It is customary to recognize three stages in the symptoms produced by chloroform. In the first stage, there is excitement, ringing in the ears, disturbed vision, and finally, loss of consciousness. The second stage is that of surgical anesthesia. The muscles are relaxed and the patient is entirely insensible to pain. The third stage is that of paralysis. It is characterized by fall of blood pressure and failure of the heart and respiration. Fibrillation of the ventricle of the heart, one of the most sudden of all forms of death, may result from light chloroform anesthesia combined with nervous excitement or stimulant drugs.

Prolonged exposure to low concentration of chloroform may lead to liver degeneration, and affection of the kidneys and pancreas. This form of poisoning may be encountered in industrial exposures. Chloroform should not be subjected to conditions which may decompose it forming phosgene and hydrogen chloride. Such conditions, include light, oxidizing substances, fire, flames. etc., towards which chloroform is less stable than most of the chlorinated hydrocarbons.

d. Ethyl Chloride:—Ethyl Chloride is decomposed in the manner similar to methyl chloride. The principal product in this case, however, is ethyl alcohol which, unlike methyl alcohol, is rapidly oxidized and does not accumulate in the body. The chief action is anesthetic and is exercised prior to the decomposition of the ethyl chloride in the body. The irritant action to the lungs is less than that of methyl chloride, and the anesthetic action is less powerful than that of chloroform. It has a depressant action upon the heart and is prone to produce fibrillation of the ventricle. The anesthetic action is rapid and recovery is usually equally rapid.

e. Ethylene Dichloride:—Ethylene Dichloride has a powerful narcotic action similar to that of chloroform and carbon tetrachloride. For single exposure of an hour or more, its toxicity appears to be of about the same order as chloroform and carbon tetrachloride, but for periods of less than an hour is less toxic than these compounds.

On the basis of animal experiments, symptoms in order of occurrence are indicated as follows: Eye and nose irritation, vertigo, static motorataxia, retching, semi-consciousness and unconsciousness accompanied by uncoordinated movements of the extremities, and death if exposure is continued. It has a distinct odor and warning symptoms are produced by relatively safe concentrations.

f. Trichloroethane:—Trichloroethane, often incorrectly called ethylene trichloride, has a strong narcotic action and its toxicity is said to be about the same as that of chloroform. It is said to cause skin lesions resembling burns so that men who are overcome by the narcotic action should have their clothing removed to avoid the irritant action on the skin. Carrieu tested trichloroethane on mice, rats, and guinea pigs, and found it to be rapidly anesthetic, causing acute congestion of the lungs and kidneys and to a lesser extent to the liver and spleen.

g. Tetrachloroethane:—Tetrachloroethane has been demonstrated as the most toxic of the aliphatic chlorinated hydrocarbons. It is not highly volatile, and acute narcosis from the fumes is never severe, but chronic poisoning is notoriously severe, and affects a fairly large portion of those exposed, the outstanding symptom being jaundice. The early symptoms begin with abnormal fatigue, general nervousness, and loss of appetite, which are followed by nausea, vomiting and dizziness. At this point, there is a progressive increase of large mononuclear cells often reaching 40 per cent; there are many immature, large, mononuclear and a slight elevation in the white cells. If exposure is continued for a period of days or even weeks, intense jaundic ensues, vomiting becomes worse, congestion, stupor, delirium, and coma usually followed by death is observed in the most severe exposures.

Wilcox in England contends that the jaundice is hepatogenous and that there is no appreciable hemolysis. This differentiates tetrachloroethane poisoning from arsine poisoning. It is distinguished from acute infectious jaundice by the absence of marked fever.

h. Pentachloroethane:—Pentachloroethane is a narcotic poisoning of considerable potency and produces toxic effects similar to those of tetrachloroethane.

i. Dichloroethylene:—Dichloroethylene is a narcotic of relatively weak action which is stated to cause fatty degeneration of the liver and kidneys if exposure is repeated and prolonged.

Autopsies have shown an excess of fat in the blood, suggesting that death from dichloroethylene poisoning was due to an embolism rather than from asphyxiation of paralysis.

j. Trichloroethylene:—Trichloroethylene is a narcotic with a potency comparable to that of chloroform and carbon tetrachloride. The acute action of a single large dose is similar to that of carbon tetra-

chloride, but the sequelae of such intoxications are not the same and there is also a decided difference in the effects of chronic poisoning. Carbon tetrachloride causes central necrosis of the liver and acute nephritis. Trichloroethylene exerts its damaging effects on the central nervous systems, with paralysis of the sensory fibers of the fifth nerve and injury to the optic nerve. Cerebral symptoms, suggestive of lesions in the capillary walls, have also been reported. It is stated that most deaths due to trichloroethylene have been from the narcotic effects of prolonged and excessive exposure without regaining consciousness. Nervous sequelae, including headache, dizziness, anorexia, and disturbed heart action have been reported as resulting from acute narcosis. Long continued exposure has been held responsible for lesions in the optic and trigeminal nerve, with complete blindness resulting in several cases. Continued exposure to trichloroethylene may finally lead to its addiction. It has been reported that men working with trichloroethylene will not inform their supervisors of illness for fear of losing their jobs, thereby losing the opportunity of inhaling this solvent and experiencing the pleasing effects which makes them feel at peace with the world.

The toxic effect of trichloroethylene has been a somewhat controversial subject. It has been stated that most of the reported injuries have prevailed in Europe, and it has been suggested that some of the observed effects may be due to impurities. However, it is agreed that this solvent does not have certain harmful physiological properties, and reasonable precautions should be exercised to prevent the continued exposure of workmen to its vapors.

k. (Tetrachloroethylene) Tetrachloroethylene, or perchloroethylene, is less narcotic than chloroform or carbon tetrachloride. When tested on dogs, it was found to have advantages over carbon tetrachloride as an anthelmintic for it was apparently not absorbed from the intestinal tract and therefore caused no injury to the liver or kidneys. However, it has been reported that inhalation of its vapors may cause slight injury to the liver and kidneys. It is usually considered to be the least toxic of industrial aliphatic chlorohydrocarbons.

l. (Vinyl Chloride) Exposure of guinea pigs to air containing vinyl chloride vapors showed symptoms which are principally those of narcosis. They range from unsteadiness and motor ataxia to complete narcosis. The principal gross pathological findings were congestion and edema of the lungs with hyperemia of the kidneys and liver.

m. (Propylene Dichloride) Studies on the toxic properties of propylene dichloride are limited. Symptoms of narcosis including headache, dizziness and vertigo have been reported. Respiratory irritation, lacrimation and anemia have also been indicated.

n. (Chloroprene) Experiments have indicated that chloroprene, the starting material for the synthetic rubber DuPrene is a toxic solvent which should be handled with the greatest precaution. Incipient poisonings may be ascertained by the determination of the icteric index, determination of albumin, reducing substances and bile pigments in the urine. Loss of weight, indigestion, and catarrhal conditions of the respiratory tract are complaints that may also indicate incipient poisonings. For a more complete summary, the reader is referred to the last abstract of this bulletin.

3. Toxic Concentrations.

a. The following table frequently quoted indicates the comparative toxicity of some of the more important chlorinated hydrocarbons. This rating must be accepted with certain reservations since various conditions such as concentration, duration of exposure, the presence of other solvents, and predisposing physical conditions may alter these toxic relations considerably, owing to differences in the character of their individual toxic action.

TABLE OF COMPARATIVE TOXICITY OF CHLORINE DERIVATIVES OF THE ALIPHATIC HYDROCARBONS.

Relative lethal concentrations by volume in air if toxicity of $CCl_4 = 1$.

Methyl Chloride	CH_3Cl*	0.6
Trichloromethane (Chloroform)	$CHCl_3$*	2.2
Tetrachloromethane (Carbon tetrachloride)	CCl_4*	1.0
Dichloroethylene	$C_2H_2Cl_2$**	1.7
Trichloroethylene	C_2HCl_3**	1.7
Perchloroethylene	C_2Cl_4**	1.6
Tetrachloroethane	$C_2H_2Cl_4$**	9.1
Pentachloroethane	C_2HCl_5**	6.2

*Waller, A. D., J.A.M.A. 53:9, 1919.

**Herrmann, G., Inaugural Dissertation; Wurzburg, C. Fuchs, 1911.

SELECTED REFERENCES

Baker, H. M.: Intoxication with Commercial Methyl Chloride. Jour. Am. Med. Assn., vol. 88, p. 1137, April 9, 1937.

Barrett, Hugh M.: The Determination of Trichlorethylene in Air. Jour. Ind. Hyg., vol. 18, no. 6, pp. 341-348, June 1936.

Barrett, H. M., Cunningham, J. G., and Johnston, J. H.: A study of the Fate in the Organism of some Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 21, no. 10, pp. 497-490, Dec. 1939.

Barrett, H. M., and Johnston, J. H.: Fate of Trichlorethylene in the Organism. J. Biol, Chem., vol. 127, pp. 765-770, March 1939.

Carpenter, Charles Patten: The Chronic Toxicity of Tetrachlorethylene. Jour. Ind. Hyg. vol. 19, no. 7, pp. 323-336, Sept. 1937.

Carrieu, W. F. A contribution to the study of Ethylene Trichloride Poisoning. Rev. d' hyg., vol. 49, pp. 349-361, May 1927.

Collier, H.: Methylene Dichloride Intoxication in Industry. A Report of Two Cases. Lancet, p. 594, March 1936.

Drinker, Cecil K:. Further Observations on the Possible Systemic Toxicity of Certain of the Chlorinated Hydrocarbons with Suggestions for permissible Concentrations in the Air of Workrooms. Jour. Ind. Hyg., vol. 21, no. 5, pp. 155-159, May 1939.

Drinker, Cecil K., Warren, Madeleine Field, Bennett, Granville A.: The Problem of possible systemic effects from Certain Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 19, no. 7, pp. 283-311, Sept. 1937.

Dyktor, H. G., and Buxell, J.: Health Hazards and Their Control in the Electroplating Industry. Ind. Hyg. Ser., City of St. Louis, Mo., p. 32, 1938.

Ebert, F.: Toxic Action of Organic Solvents and Dangers in their use. Pharm. Ztg., vol. 73. p. 854, 1928.

Eichert, H.: Trichlorethylene Intoxication. J. A. M. A., vol. 106, pp. 1652-1653, May 19, 1936.

Floret: Industrial Poisoning from Hydrocarbons of the Aromatic and Aliphated Series (Chlorinated Hydrocarbons) and its Prevention. Zentralbl. f. Gewerbehyg. N. S. vol. 4, pp. 257-261, Aug. 1927.

Frois: Tetrachlorethane Poisoning. Bull. de l'acad. d. med., vol. 88, no. 28, p. 40, July 11, 1922.

Frois: The Toxicity of Tetrachlorethane. Rev. d'hyg., vol. 44, no. 11, pp. 987-992, Nov. 1922.

Genevois, L.: Physiological Properties of Chlorinated Solvents. Ann. d'hyg., vol. 14, pp. 139-149, 1936.

Greenburg, Leonard, Mayers, May R., and Smith, Adelaide Ross: The Systemic Effects Resulting from Exposure to Certain Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 21, no. 2, pp. 29-38, Feb. 1939.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 202-228.

Harry, R. G.: The Toxicity of Industrial Solvents. Paint Manuf., vol. 6, pp. 288-290, 1936.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc. Publishers, New York, 1927.

Jordi, A.: Misuse of Trichlorethylene by Young People for Hypnosis. Experiments in a Secondary School. Schweiz. med. Wchnschr., vol. 67, pp. 1238-1240, 1937.

Kober, G., and Mayhurst, E.: Industrial Health, P. Blakiston's Son and Co., 1924, pp. 575-576, 631.

Lehman, K. B., and Schmidt-Kehl, L.: The Thirteen Most important Hydrocarbons of the Aliphatic series from the standpoint of industrial Hygiene. Kehl. Arch. f. Hyg., vol. 116, pp. 131-268, 1936.

Leri, A., and Breitel: Polyneuritis Due to Tetrachlorethane. Bull. Soc. med. d. hop., vol. 46, p. 1406, Oct. 27, 1922.

Lutz, G.: Degeneration of the Nerves Due to Chronic Poisoning from Solvents. Arch. f. Gewerbepath. u. Gewerbehyg., vol. 1, pp. 740-748, 1931.

McNally, W.: Toxicology. Industrial Medicine. Publishers, Chicago, 1937.

Martin, E.: Poisoning by Hydrocarbons. Compensation According to the French Law on Industrial Diseases (1919-1931). Med. du travail, vol. 10, pp. 151-169, 1938.

Middleton, E. L., M. D., D. P. H.: Fatal Case of Poisoning by Ethylene Chlorhydrin. Jour. Ind. Hyg., vol. 12, no. 7, p. 265, Sept. 1930.

Occupation and Health. International Labour Office, Geneva, 1934.

Ohio Department of Health, Chlorinated Hydrocarbons, 1940.

Paris Letter: Intoxications Resulting from the Manufacture of Artificial Pearls. Jour. Am. Med. Assn., vol. 80, pp. 709-710, March 10, 1923.

Parmenter, D. C., M. D.: Tetrachlorethane Poisoning and its Prevention. Jour. Ind. Hyg., vol. 2, no. 12, pp. 456-465, April 1921.

Persson, H.: Poisoning by Trichlorethylene. Acta Med. Scandinavica, Supp. 59, pp. 410-422, 1934.

Rochaiz, A.: The Danger Associated with Using Certain Plant for Chlorine Solvents in Cleaning clothes. Ann. d'hyg. puv., vol. 14, pp. 61-64, 1936.

Roholm, K.: Trichlorethylene Intoxication in Industry. Ugesk. f. Laeger, vol. 95, p. 1183, Nov. 1933.

Sartorius, F., and Boedicker, W.: On the Question of Injury to Health from Floor Cleaning Materials. Zentralbl. f. Gewerbehyg., N.S. vol. 8, pp. 103-107, April 1931.

Sharp, B. B.: Toxic Effects of Methyl Chloride Gas. Brit. Med. Jour., vol. 1, p. 336, Feb. 22, 1930.

Taylor, H.: Experiments on the Physiological Properties of Trichlorethylene. Jour. Ind. Hyg., vol. 18, no. 4, pp. 175-193, April 1936.

Tebbens, V. D.: Portable Combustion Apparatus for Field Determinations of Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 19, no. 5, pp. 204-211, May 1937.

Trillat, M.: Classifications of Trichlorethylene. Ann. d'Hyg. Pub., vol. 15, pp. 434-441, 1937.

von Oettingen, W. F.: The Halogenated Hydrocarbons: Their Toxicities and Potential Dangers. Jour. Ind. Hyg., vol. 19, no. 8, pp. 349-448, Oct. 1937.

Weber, H. H.: Methods of Analyzing Technical Solvents. VII. Arb. a. d. Reichsgesundheitsamt, vol. 72, pp. 241-243, 1937.

Welwert: Formation of Toxic Gases in the Manufacture of Washing Compounds Containing Trichlorethylene. Seifensieder-Ztg., vol. 56, p. 26, 1929.

White, John L., and Somers, Paul P.: The toxicity of Methyl Chloride for Laboratory Animals. Jour. Ind. Hyg., vol. 13, no. 8, pp. 273-275, October 1931.

Wirtschafter, Zolton T., and Schwartz, Edward D.: Acute Ethylene Dichloride Poisoning. Jour. Ind. Hyg., vol. 21, no. 4, pp. 126-131, April 1939.

CHLORINATED HYDROCARBONS: CARBON TETRACHLORIDE

Carbon Tetrachloride:

Chemical formula and synonyms—CCl_4; Perchloromethane, tetrachloromethane.

A. Principal Properties:

1. Light colorless liquid; peculiar odor; yielding heavy vapors; non-inflammable; poisonous; Sp. Gr. 1.5944 at 20°C. Miscible with alcohol, ether, chloroform, benzol, benzine and most of the fixed and volatile oils. Very slightly soluble in water. Wt. per liter of vapor 6.39 gr.

B. Principal Industrial Uses:

1. Solvent; fire extinguishers; cleaning compounds; chloroform manufacture; chlorinating organic compounds; substitute for oil in electrical transformers and high tension switches; solvent for fats and oils; electroplating; metal polishes; lacquers; extracting perfumes from flowers; paraffin solvent; recovering waxes and resins from raw materials; rubberizing fabrics; rubber cements; manufacture of gelatinous water-soluble soaps from sulfinated oils and resins; degreasing textiles.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation.
   b. Ingestion.

2. Symptoms
   a. *Acute.* Loss of appetite, nausea, vomiting, cough, headache, somnolence, nervousness, marked excitement, mental confusion, vertigo, increased pulse and respiration, air hunger, weakness, burning sensation in the epigastrium, diarrhea, loss of weight, jaundice, secondary anemia, pain and tenderness over an enlarged liver, and tendency to intestinal hemorrhages. Visual disturbances include blurred vision, color confusion and disturbance of near vision. The urine shows increased acidity, increased phosphates, casts and albumin. There is suppression of urine, uremia, coma, convulsions, and death. At autopsy is found central necrosis and fatty degeneration of the liver and kidneys.
   b. *Chronic.* Prolonged exposure affects persons differently; those with cerebral symptoms, those with liver and kidneys affected, those with kidneys only attacked, those where the lungs are irritated and edematous, and those with inflamed skins.

3. Laboratory Procedure
   a. Increased icterous index.
   b. Urinalysis—Albumin and casts usually present.

4. Pathology
   a. Autopsy findings of death caused by carbon tetrachloride poisoning:
      (1) Scattered small hemorrhages in serosal coverings of abdominal viscera, in lungs and pancreas.
      (2) Liver revealed extensive degeneration with removal of degenerated cells centrally and definite effort at regeneration in peripheral ascinal areas extending as tubule-like strands toward the centers.
      (3) Nephritic changes: Distension of the spaces of Bowman with albuminous precipitate; by swelling of the lining cells, and swelling and vacuolation of the cells of the proximal convoluted tubules; by degeneration and necrosis of the cells of the distal convoluted tubules, and of those of the loops of Henle, with desquamation; and by the presence of granular, hyaline and cellular casts in the tubules, with plugging of their lumens.

5. Toxic Concentrations
   a. The concentration of carbon tetrachloride vapors should not exceed 100 parts per million where individuals are exposed for a prolonged period of time.

D. **Sanitary Corps Officer:**

1. Adequate ventilation and control thereof.
2. Avoidance of closed-in areas for its use and high temperatures.
3. Persons using it as a fire extinguisher should be protected with gas masks.
4. All vessels containing carbon tetrachloride or mixtures of which it is a constituent should be labelled "dangerous" and all accidents resulting from it should be reported.

E. **Medical Control.**

1. Preemployment placement examination
   a. All those persons having evidence of nephritis, diabetes, myocardial degeneration, high blood pressure or those using alcohol, should not be exposed to carbon tetrachloride.
2. Medical
   a. The recognition and diagnosis of carbon tetrachloride poisoning.
3. Treatment
   a. The administration of glucose and calcium has been found to be helpful symptomatic treatment.

SELECTED REFERENCES

Aubertin, E., Lacoste, A., de Lachaud, R., and Martinet, R.: Examinations of Liver Functions in Dogs Poisoned with Carbon Tetrachloride and Phosphorus Oil. Compt. rend. Soc. de biol., vol. 127, pp. 57-60, 1938.

Barrett, H. M., McLean, D. L., and Cunningham, J. G.: A comparison of the Toxicity of Carbon Tetrachloride and Trichloroethylene. Jour. Ind. Hyg. Toxicol., vol. 20, pp. 360-79, 1938.

Barrett, H. M., McLean, D. L., and McHenry, E. W.: Experiments with an "Antinecrotic" Material Prepared from Liver. Jour. Pharmacol. Exp. Ther., vol. 64, pp. 131-137, 1938.

Bollman, J. L.: Experimental Cirrhosis: Relation of Hepatic Damage and Plasma Proteins to Ascites. J. A. M. A. (Soc. Proc.), vol. 110, p. 842, Mar. 12, 1938.

Boveri, M. P.: Occupational Poisoning by Carbon Tetrachloride. Med. du travail., pp. 280-283, July 1930.

Briese, E.: The Effect on the Blood Cells of the Fetal Rat Produced by the Inhalation of Carbon Tetrachloride by the Mother During Gestation. Am. Jour. Med. Sci., vol. 195, pp. 787-793, June 1938.

Burrage, L. J., and Allmand, A. J.: The Effect of Moisture on the Sorption of Carbon Tetrachloride from an Air Stream by Activated Charcoal. Jour. Soc. Chem. Ind., vol. 57, pp. 424-31, cf.

Davis, G. G., and Hanelin, H. A.: Carbon Tetrachloride Poisoning. Indust. Med., vol. 6, pp. 24-29, 1937.

Dudley, S. F.: Toxic Nephritis Following Exposure to Carbon Tetrachloride. Jour. Ind. Hyg., vol. 17, pp. 93-110, 1935.

Erickson, C. C., Heckel, G. P., and Knutti, R. E.: Blood Plasma Proteins as Influenced by Liver injury induced by carbon Tetrachloride and Gum Acacia. Am. Jour. Path., vol. 14, pp. 537-556, Sept. 1938.

Graham, W. H.: Simulation of the "Acute Abdomen" In Carbon Tetrachloride Poisoning. Lancet., pp. 1159-60, May 21, 1938.

Hamilton, A.: Industrial Toxicology. Harper & Bros. Publishers, New York, 1934, pp. 205-211.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 158-161.

Kehrer, J. K. W., and Oudendal, A. J. F.: Poisoning with Carbon Tetrachloride Abstr. as follows from Nederl. Tijdschr. v. Geneesk., vol. 70, pp. 746-757, 1926.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., Publishers, Philadelphia, 1924.

Loewy, I.: Chronic Carbon Tetrachloride Poisoning. Arch. f. Gewerbepath., vol. 6, pp. 157-159, 1935.

McNally, W.: Industrial Solvents. Industrial Medicine, 1, 296, 1938.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 693-707.

Moller, K. C.: Carbon Tetrachloride Poisoning In Shampooing and Dry Cleaning. Jour. Ind. Hyg., vol. 15, pp. 418-432, 1933.

Occupation and Health. International Labours Office, Geneva, 1930, pp. 370-372.

Ohio Department of Health, Carbon Tetrachloride, 1940.

Peery, T. M.: Carbon Tetrachloride Poisoning in Man. Stages of Hepatic Damage and Repair in Man. Arch. Path., vol. 26, pp. 923-41, 1938.

Radhakrishna Rao, M. V.: Cirrhosis of Liver Following Chronic Intoxication with Carbon Tetrachloride: Experimental Study. Indian Jour. Med. Res., vol. 23, p. 1007, Apr. 1936.

Robbers, H.: Three Non-Fatal Cases of Poisoning from the Moth Destroyer "Melan" (P-Dichlorbenzene and Carbon Tetrachloride). Samm. v. Vergift., vol. 9, pp. 37-40, 1938.

Schutz, H.: Hepato-Renal Syndrome in Carbon Tetrachloride Poisoning. Arch. f. Gewerbepath., u. Gewerbehyg., vol. 8, pp. 469-500, 1938.

Smyth, H. F.: Carbon Tetrachloride in Industry. The Present Status and Plans for Further Necessary Studies. Indust. Med., vol. 4, pp. 12-15, 1935.

Smyth, Henry Field, Smyth, Henry F. Jr., and Carpenter, Charles P.: The Chronic Toxicity of Carbon Tetrachloride: Animal Exposures and Field Studies. Jour. Ind. Hyg. and Toxicology, vol. 18, no. 5, pp. 277-298, 1936.

Starr, E. B.: Carbon Tetrachloride Cement, Poisoning by, with References to Early Symptoms of Benzol Poisoning. Jour. Ind. Hyg., vol. 4, pp. 203-21, 1922-1923.

Stewart, H. L., and Cantarow, A.: Experimental Carbon Tetrachloride Poisoning in the Cat. II. The Influence of Ligation of Single Bile Ducts. Arch. Path., vol. 26, pp. 1121-1130, 1938.

Tabasaka, T.: Pulmonary Hemorrhages in Acute Carbon Tetrachloride Poisoning. Abstr. as follows from Deutsch. Ztschr. f. d. ges. gerichtl. Med., vol. 11, no. 5, in Munchen, med. Wchnschr., vol. 73, p. 503, Mar. 19, 1926.

Vallery-Radot, P., Maurice, G., Domart, A., and Gauthier-Villars: Kidney and Liver damage from Inhalation of Carbon Tetrachloride by Rabbits. Compt. rend. Soc. de Biol., vol. 128, pp. 482-483, 1938.

Yanagihara, D.: Amount of Phenol in the blood in Experimental Poisoning by carbon Tetrachloride, chloroform and Phosphorus and in Mechanical injury to the Liver. Jour. Chosen Med. Assoc., vol. 29, pp. 37-57, Jan. 20, 1939.

Zolezzi, G.: Organic Lesions following upon Intoxication by carbon Tetrachloride. Medicina Lavoro., vol. 28, pp. 289-301, 1937; Chimie and industrie., vol. 39, p. 888.

CHLORINATED DIPHENYLS

Chemical Formula and Synonyms:

(Chloronaphthalene) $C_{10}H_7Cl$, naphthyl chloride.
(Dichloronaphthalene) $C_{10}H_6Cl_2$.
(Trichloronaphthalene) $C_{10}H_5Cl_3$.
(Tetra-, penta-, and hexa-chloronaphthalene) $C_{10}H_4Cl_4$, $C_{10}H_3Cl_5$, $C_{10}H_2Cl_6$.
(Chlorodiphenyl) C_6H_5-C_6H_4Cl, chlorobiphenyl.
(Dichlorodiphenyl) $(Cl\text{-}C_6H_4)_2$.

A. Principal Properties:

1. Chloronaphthalene
   a. (Alpha) Sp. Gr. 1.194 at 20/4°C.
   b. Soluble in alcohol and ether, insoluble in water.
   c. (Beta) Sp. Gr. 1.266 at 16°C.
   d. Very soluble in alcohol and ether, insoluble in water.
2. Dichloronaphthalene
   a. There are ten isomers of this compound with properties ranging as follows: Sp. Gr. 1.292-1.315. As a group they are soluble in alcohol and ether and insoluble in water.
3. Trichloronaphthalene
   a. (1, 4, 5) m.p. 131°C. Very soluble in hot alcohol. (1, 4, 6) m.p. 65-6°C., slightly soluble in hot alcohol.
4. Tetra-, penta-, and hexa-chloronaphthalenes
   a. The properties of these groups of naphthalenes are similar to those of the lower chlorinated naphthalenes and are not discussed individually.
5. Chlorodiphenyl. (Crystals)
   a. Insoluble in water, soluble in ligroin.
6. Dichlorodiphenyl
   a. Insoluble in water, very soluble in alcohol and ether (4, 4′). Sp. Gr. 1.439; m.p. 148°C., b.p. 315-9°C. Insoluble in water.

B. Principal Industrial Uses:

1. Chlorinated naphthalenes and diphenyls are used in various proportions as an insulating wax for electrical equipment.

C. Systemic Poisoning:

1. Modes of entry
   a. Inhalation of vapors.
   b. Possible absorption through skin.
2. Symptoms
   a. Systemic—Symptoms most frequently encountered are weakness, nausea, anorexia, vertigo, progressive jaundice, and pigmentation of the skin. In fatal cases, the individual may become incoherent and delirious, and dies in a coma.
   b. Skin—The most common effect is a characteristic skin eruption usually located about the face, forehead, back, and buttocks. These are usually acneform eruptions characterized by pustules, papules and comedones. The association of comedones in large numbers, with a severe itching, has given it the name of "blackhead itch" among the workers. The eruptions may involve the deeper layers of the skin and result in permanent scarring. In severe cases, there is a tendency to secondary infection. A furunculosis of the cheek superimposed on an acneform eruption resulting in death from septicemia has been reported.

3. Pathology

a. The characteristic pathological findings at autopsy are associated with the liver. Extensive necros fibrosis, regeneration of the liver with a typical picture of subacute yellow atrophy is revealed.

4. Toxic Concentrations

a. On the basis of observations made on a series of animals Drinker has set up limits of permissible con centrations for the various chlorinated naphthalene preparation.

b. A list of 14 chlorinated Hydrocarbons, with Chlorine Contents and Permissible Limits (in Mg./Cu.M.) for the air in workrooms.

Compound	*Chlorine Content*	*Permissible Limit*
	%	mg./cu.m.
1. Trichloronaphthalene plus a trace of tetrachloronaphthalene. Tested upon rats by inhalation and by feeding	49.9	10.0
2. Tetra and pentachloronaphthalenes. Tested upon rats by inhalation and by feeding	56.4	1.0
3. Penta and hexachloronaphthalenes. Tested upon rats by inhalation and by feeding, and upon dogs by feeding alone	62.6	0.5
4. Tetra and pentachloronaphthalenes plus refined chlorinated diphenyl. Tested upon rats by feeding	43.5	0.5
5. 90% penta and hexachloronaphthalenes plus 10% chlorinated diphenyl benzene. Tested upon rats by inhalation and by feeding	63.0	0.5
6. Chlorinated diphenyl plus chlorinated diphenyl benzene. Tested upon rats by inhalation and by feeding	65.0	0.5
7. Chlorinated diphenyl oxide. Tested upon rats by inhalation	54.0	0.5
8. Chlorinated diphenyl oxide. Tested upon rats by inhalation	57.0	0.5
9. Chlorinated diphenyl. Tested upon rats by inhalation	50-55	0.5
10. Hexachlor diphenyl oxide plus 5% trichloronaphthalene. Tested upon rats by inhalation	50-55	0.5
11. Hexachloronaphthalene and crude chlorinated diphenyl. Tested upon rats by inhalation	Un-	0.5
12. Special chlorinated naphthalene. Tested upon rats by inhalation	50-56	0.5
13. Chlorinated diphenyl. Tested upon rats by inhalation	68	10.0
14. Chlorinated diphenyl benzene. Tested upon rats by inhalation	60	0.5

D. Sanitary Corps Officer:

1. Proper control measures.

E. Medical Control:

1. Pre-employment—Those who may have previous liver damage. Such conditions include any previous liver disease, arsphenamine treatment for syphilis, pregnancy, and recent general anesthesia.

SELECTED REFERENCES

Drinker, Cecil K.: Further Observations on the Possible Systemic Toxicity of Certain of the Chlorinated Hydrocarbons with Suggestions for Permissible Concentrations in the Air of Workrooms. Jour. Ind. Hyg., vol. 21, no. 5, pp. 155-159, May 1939.

Greenburg, Leonard, Mayers, May R., Smith, Adelaide Ross: The Systemic Effects Resulting from Exposure to Certain Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 21, no. 2, pp. 29-38, Feb. 1939.

McNally, W.: Toxicology Industrial Medicine, Publishers, Chicago, 1937, p. 1005.

Mayers, May R., and Silverberg, Mabel G.: Skin Conditions Resulting from Exposure to Certain Chlorinated Hydrocarbons. Jour. Ind. Hyg., vol. 20, no. 3, pp. 244-258, March 1938.

Ohio Department of Health, Chlorinated Naphthalenes and Diphenyls, 1940.

Yaglou, C. P., Sands, F. W., and Drinker, Philip: Ventilation of Wire Impregnating Tanks using Chlorinated Hydrocarbons. Jour. Ind. Hygiene, vol. 20, no. 6, pp. 401-418, June 1938.

CHLORINE ACID

Chemical Formula and Synonyms:

(Chlorine) Cl_2, liquid bleach.
(Hydrochloric Acid) HCl Chlorohydric acid; hydrogen chloride; muriatic acid.

A. Principal Properties:

1. Chlorine
   a. Heavy, greenish-yellow gas or liquid, poisonous. Sp. gr. 2.491; m.p. –102°C.; b.p. –33.6°C. Soluble in water and alkalis. Weight per liter, 2.95 gr.
2. *Hydrochloric Acid*
   a. Hydrochloric acid, which is a solution of hydrogen chloride in water, is prepared commercially in different strengths and degrees of purity. Common impurities are iron, sulfuric acid and arsenic. The specific gravity of a 35.37% hydrogen chlorine acid is 1.178-1, 188. Hydrogen chloride is a colorless gas; sp. gr. 1.268 (air), m.p. –111°C.; b.p. –85°C., and is soluble in alcohol, water and ether.
3. Important Compounds
   a. Sodium chloride, calcium chloride, hydrochloric acid, perchloric acid and many other inorganic compounds of chlorine; chloroform, carbon tetrachloride, chlorobenzene, chlorophenols and many other organic compounds of chlorine.

B. Principal Industrial Uses:

1. Chlorine
   a. Organic synthesis; chlorination; hydrochloric acid; textile bleaching; liquor; water purification; military poison gas; metallurgy (recovery of gold and silver from their ores, extraction of copper, lead and zinc from mixed ores, separation of tungsten and vanadium from their ores; detinning scrap white cast iron and dezincing scrap galvanized iron obtaining, as by-products, tin and zinc chlorides, respectively, free of iron, when using gaseous chlorine absolutely free of water); inks, paper (bleaching); rubber substitutes; chlorinated rubber, water purification.
2. Hydrochloric Acid
   a. Chrome tanning; leather industry; organic synthesis; dye manufacture; dyeing; artificial silk manufacture; intermediates; bleaching of edible and technical fats and oils; metallurgy (etching; galvanizing metals, recovery of zinc from galvanized scrap, solvent for gold (with nitric acid) and silver, pickling iron for tinning purposes, electroplating, wire manufacture, purifying iron ores, metallurgy of copper and galena containing zinc); photography; paint pigments; glue and gelatin manufacture; paper manufacture; process engraving and lithographing; purification of soap stock; textiles (bleaching, dyeing, printing, mercerizing); ink manufacture; tinning and soldering; reclaimed rubber; sugar (purification of Charcoal, diffusor auxiliary in beet sugar manufacture); ceramics, glass purification of sand and clay; analytical reagent.

C. Systemic Poisoning:

1. Modes of entry
   a. Inhalation.
   b. Ingestion Accidental or suicide attempts.
2. Chlorine
   a. The symptoms of poisoning by chlorine gas are primarily concerned with irritation of the respiratory system. Fifteen parts per million causes immediate irritation of the throat and with higher concentration irritation of the conjunctiva. Fifty parts per million produces strong dyspena, cyanosis and death. The symptoms will vary with the concentration and time during which the gas is inhaled. Sustained exposures may cause bronchitis, bronchiectasis, loss of sense of smell, loss of appetite with loss of weight, headache, giddiness, insomnia and cardiac disturbances.

The following table indicates the effect of exposure to various concentrations of chlorine:

TOXIC CONCENTRATIONS

	Parts of Chlorine per Million Parts of Air
Least detectable odor*	3.5
Least amount causing immediate irritation to the throat*	15.1
Least amount causing coughing*	30.2
Maximum concentration allowable for prolonged exposure*	1.0
Maximum concentration allowable for short exposure (½ to 1 hour)**	4.0
Dangerous for even short exposure	40 to 60
Rapidly fatal for short exposure**	1000

D. **Symptoms:**

1. Hydrochloric Acid

a. Hydrochloric acid poisoning, according to McNally, is more common than by sulphuric or nitric acid acid (not including oxides of nitrogen). The clinical symptomatology in man includes irritation of mucous membranes, conjunctivitis, pharyngeal, laryngeal and bronchial catarrh, and dental caries. The occasional occurrence of unusually dry hydrochloric acid gas under some conditions of atmospheric heat and absence of moisture explains the increased degree of toxicity which this gas sometimes exhibits.

As previously mentioned, the possibility of the production of the very poisonous gas, arseniureted hydrogen or arsine, should not be overlooked whenever metals are treated with hydrochloric or sulfuric acid, as in the "pickling of iron" or the stripping of galvanizing by means of acid. Either when an acid acts upon a metal with the evolution of nascent hydrogen in the presence of a soluble compound of arsenic. Such conditions are common in industry and the poisonings resulting may mistakenly be attributed to acid fumes.

Poisoning by ingestion of hydrochloric acid is frequently the result of suicidal attempt. Accidental ingestion is not so frequent because of the strong irritative action of the acid. Concentrated acid introduced into an empty stomach produces grave symptoms almost immediately. Burns of the lips, tongue and throat are at first white, later becoming dark brown. Acute laryngeal irritation may occur. Pain is instantaneous and affects the mouth, throat and abdomen. Vomiting ensues and there is great weakness with feeble pulse. Anxiety and dyspnea are present. Absorption is evidenced by nervous symptoms such as spasms, dilation of pupils and fainting. Patients surviving the acute symptoms are subject to bronchitis and pleurisy from inhalation of the fumes and may develop strictures of the esophagus and stomach.

PHYSIOLOGICAL RESPONSE OF MAN TO VARIOUS CONCENTRATIONS

Acute Poisoning

Milligrams per Liter	*Parts per Million*	*Length of Exposure*	*Effect*
0.05	35	10 minutes	Sneezing, bronchial irritation, choking sensation.
1-2	670-1250	After few minutes	No longer bearable even by acclimatization.

Chronic Poisoning

0.013	9	6 hours	No important symptoms. (Lehmann.)
0.01	6.5	Several hours	Troublesome. (Hess.)
0.06-0.13	40-90	½-1 hour	Without consequences. (Lehmann.)
1.5-2.0	1000-1350	½-1 hour	Dangerous. (Hess.)
1.84-2.6	1250-1750	½-1 hour	Immediately or subsequently fatal. (Lehmann.)

*Ficklen, Joseph B.: Manual of Industrial Health Hazards. Service To Industry, West Hartford, Conn., 1940; 102-103.

Standard[1,2,3,4]			
Milligrams per Liter	*Parts per Million*	*Length of Exposure*	*Comment*
0.015	10	Work Period	Maximum allowable concentration.

E. Sanitary Corps Officer:

1. No liquid chlorine should be discharged from any container unless the person in charge of the operation has the requisite knowledge and experience. Cylinders and drums containing liquid chlorine should be properly stored (under cover, not in the main building, protected from heat and damp, away from danger of fire or explosion, and in a place from which they can readily be removed in case of fire). In opening a chlorine cylinder the supplier's directions should be strictly followed. Containers should be so secured that all risk of unexpected movement during discharging is avoided. Heat should on no account be applied to the containers to assist liberation of the gas. Precautions should be taken to avoid damage to valves, pipes and couplings. Illustrations are given of suitable slings for lifting cylinders and drums, and such slings should be tested to twice the required tension. Wrought iron slings should be annealed and examined periodically. It is essential that compressed air for use with liquid chlorine in tank wagons and storage tanks should be perfectly dry and free from lubricating oil, otherwise corrosion of the tank will occur. If the use of containers in a confined space is unavoidable efficient exhaust fans working at low level should be provided. Leakages can be traced by means of ammonia fumes. When a container leaks the point of leakage should be turned upwards to prevent leakage of liquid. The precautions to be taken in the event of a substantial escape of gas occurring are outlined. Canister respirators should be provided and stored in convenient places, and where large quantities of liquid chlorine are stored there should be a self-contained oxygen breathing apparatus, half-hour type, at each end of the plant, and workmen should be trained in its use.

F. Medical Control

1. Treatment
   - *a.* Expectorants
   - *b.* Venesection
   - *c.* Oxygen
   - *d.* Stimulants
   - *e.* Treat for Shock

SELECTED REFERENCES

Barbour, H. G., Hjort, A. M., and Taylor, F. A.: Drugs After Chlorine gassing. I. The Influence of Morphine Upon the Fatality of Chlorine Poisoning. Jour. Pharmacol. and Exper. Therap., vol. 14, no. 1, pp. 55-59, Sept. 1919.

Barbour, H. G.: Drugs after Chlorine Gassing. II. Observations Upon the Treatment of gassed dogs with circulatory Stimulants. Jour. Pharmacol. and Exper. Therap., vol. 14, no. 1, pp. 61-64, Sept. 1919.

Barbour, H. G. and Williams, H. W.: The Effects of Chlorine Upon Isolated Bronchi and Pulmonary Vessels. Jour. Pharmacol. and Exper. Therap., vol. 14, no. 1, pp. 47-53, Sept. 1919.

Etienne-Martin, P.: Poisoning by Chlorine. Med. du Travail, vol. 4, pp. 83-96; 138-152, 1932.

Ficklen, Joseph B: Manual of Industrial Health Hazards. Service to industry, Publishers, 1940, pp. 79-80; 102-104.

Flury, F., and Zernik, F.: Schadliche Gase. J. Springer, Berlin, 1931, pp. 117-121, 125-128.

Freund, R.: Working with Compressed Chlorine Gas in Practice. Chem.-Ztg., vol. 52, pp. 33-34, 1928.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 6-8.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 87, 126-127; 131-132.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 650; 652.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 69-74; 111-115.

Memorandum on Precautions in the Handling, storage and use of liquid Chlorine. Factory Department, Home Office, Form 1868, H. M. Stationery Office, London, April 1937.

Nicolas, J., and Lacassagne, J.: A Case of Chlorine Acne. Bull. Soc. franc. de dermat, et syph., p. 223, March 1929.

Nielsen, J. Behn: Behavior of Gas-Mask Charcoal Towards Phosgene and Chlorine. Z. ges. Schiess-Sprengstoffw., vol. 27, pp. 280-284, 1932.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 429-436 ; 989-993.

Ohio Department of Health, Chlorine and Hydrochloric Acid, 1940.

Reyerson, L. H., and Wishart, A. W.: The Sorption of Chlorine by Activated Charcoal. Jour. Phys. Chem., vol. 42, pp. 679-685, 1938.

Rochaix, A.: The Dangers Associated with using certain plant for Chlorine Solvents in cleaning clothes. Ann d'hyg., pub. vol. 14, pp. 61-64, 1936.

Safe Handling of Chlorine: Safe Practices Pamphlet No. 71, Nat. Safety News, vol. 13, pp. 39-43, March, 1926.

Shera, Vrian L.: Safe and Proper Handling of Liquid Chlorine. Pacific Chem. Med. Inds., vol. 2, no. 5, pp. 19-22, 1938.

Sklanskaja, R. M., Klaus, L. M., Sidorovau, L. M., and Raporort, J. L.: Influence of Chlorine on the Female Organism. Gigiena truda i teknika bezopasnosti, no. 1, pp. 12-25, 1935.

Tupholme, C. H. S.: Safety Hints for Liquid Chlorine. Chem. Industries, vol. 43, p. 20, 1938.

Walton, D. C., and Eldridge, W. A.: The Action of Chlorine on Men Poisoned by Toxic smokes. Jour. Pharmacol. and Exper. Therap., vol. 35, pp. 241-256, 1929.

Warson, T. E.: Note on the Determination of Chlorine. Jour. Am. Water Works Assoc., vol. 29, pp. 1775-1776, 1937.

Yoe, J. H.: A Convenient Apparatus for the Determination of Low Concentrations of Chlorine in Chlorine-Air Mixtures. Jour. Lab. and Clin. Med., vol. 10, pp. 1041-1045, 1925.

CHROMIC ACID: RELATED COMPOUNDS

A. **Principal Properties:**

1. Hard, lustrous, silvery-white, non-magnetic metal.
2. Not oxidized at ordinary temperatures.

B. **Principal Industrial Uses:**

1. Metal has little use except in certain alloys.
2. Refractory brick and furnace linings-chromite (FeO, Cr_2O_3).
3. Dyes, inks, pigments, photography-chromium compounds.
4. Chromium plating—chromic acid (CrO_3) principal source of industrial poisoning.

C. **Chrome Poisoning—systemic poisoning by chromium compounds has not been clearly shown.**

1. Modes of entry.
   a. Contact with skin and mucous membranes.
   b. Inhalation.
2. Symptoms and Diagnosis.
   a. Painful, pit-like, phagedenic slow healing ulcers skin and mucous membranes.
   b. Inflammation and perforation of cartilaginous portions of nasal septum.
   c. Eczematous eruptions.
   d. Conjunctivitis.
   e. Irritation of respiratory passages.
   f. May result in inflammation of pulmonary tissue.
   g. Pulmonary cancer from chrome dust has not been clearly demonstrated.
3. Toxic Concentration:
   1 mg. chromium dust or vapor per ten cubic meters (353 cu. ft.) of air.

D. **Instructions to Operators:**

1. Operators should guard against injury to the nasal tissues by applying vaseline to them several times daily.
2. Rubber boots, gloves and aprons should be used when feasible to prevent contact of chromic acid with any abraded skin; or facilities provided for washing hands frequently and protective ointment applied to hands.

E. **Sanitary Corps Officers:**

1. Frequent checks to ascertain proper ventilation and efficiency of exhaust methods.
2. Determination of chromium dust or vapor per ten (10) cubic meters of air.

F. **Medical Control:**

1. Periodic check every three months of employees exposed to chrome and its compounds.
2. Treatment of chrome ulcers should include washing with thiosulphate solution, application of ointment and a waterproof covering applied.

G. **Suggested Chrome Hazard Examinations:**

1. Name, Address, Age, Race, Sex, Number of Years Employed as Chrome Plater, Hours per Day and Hours per Week.

2. History of Previous Exposure to Chrome.

3. History and Physical and Dates.

   a. Present Occupational History:

      (1) Wear gloves

      (2) Wear mask

      (3) Wash and bathe daily

      (4) Anoints face, hands, nostrils with vasoline or oil

      (5) Change clothing in plant

   b. Personal History:

      (1) Any operation on nose

      (2) Any nasal trouble

      (3) Any nasal trouble since working in plating room

      (4) Nose bleed

      (5) Appetite

      (6) Loss of weight

      (7) Throat trouble

      (8) Digestive disorders

   c. Physical Examination—Skin Condition:

      (1) Ulcer of skin

      (2) Open wounds

      (3) Eczema

   d. Nasal Examinations:

      (1) Septum perforated

      (2) Ulcers

      (3) Inflammation

      (4) Bloody Mucosa

   e. General Chest Findings:

   f. Remarks:

SELECTED REFERENCES

Belgian Letter: Occupational Diseases Among Workers in the Chromates. Jour. Am. Med. Assn., vol. 109, p. 1922, Dec. 4, 1937.

Blair, J.: Chrome Ulcers. Report of Twelve Cases. Jour. Am. Med. Assn., vol. 90, pp. 1927-1928, June 16, 1928.

Blair, J.: Health Hazards in Chromium Plating. Ohio State Med. Jour., vol. 27, p. 142. Feb. 1931.

Bloomfield and Blum: Health Hazards in Chromium Plating. Public Health Reports Sept. 7, 1928, pp. 2330-2347.

Chrome Plating and Anodic Oxidation: Reprinted from Form 1891, April 29, 1930, Home Office, London.

Foreign Letter (Japan): Poisoning from Chromium. Jour. Am. Med. Assn., vol. 89, p. 2055, Dec. 10, 1927.

Gerbis: The Protection of the Nasal Septum from Corrosive Action of Inhaled Chromates. Zentralbl. f. Gewerbehyg., N. S. vol. 1, pp. 10-11, July 1924.

Goldman and Riley: Control of Chromic Acid, Mists from Plating Tanks, Public Health Reports, vol. 52, No. 6, February 5, 1937.

Gorodetskil, G. A., and Makhover, S. L.: Determination of Small Quantities of Chromium in the Air of Industrial Plants. Hig. Truda 15, no. 1, pp. 84-85, 1937; Chimie and industrie, vol. 38, p. 889.

Hamilton, A.: Industrial Toxicology. Harper and Bros., Publishers, New York, 1934, pp. 17-19.

Industrial Poisons Committee, Chemical Section, National Safety Council: Chromium. Safe Practice Sheet No. 3, p. 63, Oct. 1925.

Industry Rep., Retail Credit Co.: Chromium Plating, vol. 4, pp. 1-6, July 23, 1932.

Joules, H.: Asthma from Sensitization to Chromium. Lancet, pp. 182-183, July 23, 1932.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., Publishers, Philadelphia, 1924, pp. 588, 651, and 847.

Labat, J. A.: Toxicology and Hygiene of Chromium. Bull. trav. soc. pharm. Bordeaux, vol. 76, pp. 191-201, 1938.

Lapin, N. P., Warganow, W. F., Druskin, S. L., Worochobin, I. G., and Sapolsky, W. W.: Industrial Hygiene Measures in Chromium Plating. Gigiena Truda i tekhnika bezopasnosti., no. 5, pp. 51-55, 1934.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 126-129.

Mauro, V.: Cachexia and Hematological Alterations in Experimental Chromium Poisoning. Rass. Med. all. al Lavoro Ind., vol. 6, pp. 435-440, 1936.

Mikhal' chishin, G. T.: Drop Method for an Approximate Determination of Chromium. Univ. etat kiev, Bull. sci., Rec. Chim. no. 3, pp. 85-89, 1937.

Occupation and Health. International Labour Office, Geneva, 1930, pp. 437-448.

Ohio Department of Health, Chromic Acid and Its Derivatives, 1940.

St. Galloro: Experimental Investigation on Occupational Chromium Poisoning in the Electro-Chromium plating industry. Folia med., vol. 24, pp. 1256-1265, 1938.

Shaverdova, E. I.: Protector Liquid in Electrolytic Chromium Plating Gigiena truda i tek. bezopasnosti., no. 1, pp. 89-91, 1937.

van der Walt, C. F. J., and van der Merwe, A. J.: Colorimetric Determination of Chromium in Plant Ash, Soil, Water, and Rocks. Analyst., vol. 63, pp. 809-811, 1938.

Verkhovskaia, P. I.: Use of Protector Liquids in Chromium Plating. Gigiena truda i tek. bezopasnosti., no. 2, pp. 33-36, 1937.

CYANIDES

Chemical Formula and Synonyms:

(Hydrocyanic acid) HCN, hydrogen cyanide, prussic acid, formonitrile.
(Sodium cyanide) NaCN.
(Potassium cyanide) KCN.
(Cyanogen) $(CN)_2$.
(Cyanogen chloride) ClCN, chloride of cyanogen.
(Cyanogen bromide) BrCN, bromide of cyanogen.
(Cyanogen iodide) ICN, iodine of cyanogen, iodine cyanide.
(Copper cyanide) $Cu(CN)_2$, cupric cyanide.

A. **Principal Properties:**

1. Hydrocyanic acid
   a. Colorless gas appearing in commerce in aqueous solutions (generally 2% or 10%); colorless liquid; vapors intensely poisonous; odor of bitter almonds. Sp. gr. (gas) 0.697.
   b. Soluble in water, alcohol, and ether. Weight per liter of gas, 1.12 gr.
2. Sodium cyanide
   a. White, deliquescent, crystalline powder; exceedingly poisonous.
   b. Soluble in water; slightly soluble in alcohol.
3. Potassium cyanide
   a. White, amorphous, deliquescent lumps or crystalline mass; faint odor of bitter almonds; extremely poisonous, do not handle with bare hands. Sp. gr. 1.52 at 16°;
   b. Soluble in water, alcohol, and glycerol.
4. Cyanogen
   a. Colorless gas; pungent penetrating odor; burns with a purple-tinged flame; extremely poisonous. Sp. gr. 1.8064 (compared to air):
   b. Soluble in water, alcohol, and ether. Weight per liter, 2.16 gr.
5. Cyanogen chloride
   a. Colorless liquid; poisonous. Sp. gr. 1.2.
   b. Soluble in water, alcohol, and ether. Weight per liter, 2.56 g.
6. Cyanogen bromide
   a. Needles. Sp. gr. 2.015 at 20.4°C.
   b. Soluble in alcohol, ether, and water; poisonous. Weight per liter of vapor, 4.40 g.
7. Cyanogen iodide
   a. Colorless needles; very pungent odor; acrid taste; violent poison.
   b. Soluble in hot water; very soluble in alcohol and ether.
8. Copper cyanide
   a. Green powder; exceedingly poisonous. Keep well stoppered. Soluble in acids and alkalis; insoluble in water.

Decomposes when dried to form cyanogen and cuprous cyanide.

B. **Principal Industrial Uses:**

1. Hydrocyanic acid
   a. Chemical analysis; medicine (very dilute solution as cough sedative and for nervous stomach) insecticide (gassing citrus trees); organic synthesis; military poison gas.
2. Sodium cyanide
   a. Extraction of gold and silver from ores; electroplating; heat treatment of metals; making hydrocyanic acid; insecticide.

3. Potassium cyanide
   a. Extraction of gold and silver from ores; electroplating; heat treatment of steel; reagent in analytical chemistry; insecticide; fumigant; reagent in manufacture of various intermediate organic cyanogen derivatives; paper manufacture; pharmaceutical preparations; fixative in photography; process engraving and lithography; fumigant for raw cotton; fumigant for grain elevators; fumigant for citrus fruits.
4. Cyanogen
   a. Organic syntheses; poison gas in warfare.
5. Cyanogen chloride
   a. Organic synthesis; manufacture of military poison gas.
6. Cyanogen bromide
   a. Extracting gold from certain refractory ores.
7. Cyanogen iodide
   a. Taxidermists' perservative.
8. Copper cyanide
   a. Metallurgy; intermediates (introduction of the cyanide group in place of the amino radical in aromatic organic compounds).

C. Poisoning—Systemic

1. Modes of entry
   a. All channels, even through the unbroken skin and mucous membranes. The most important path of entry of the gaseous vapors is by the respiratory tract.
2. Symptoms
   a. *General*—Cyanide compounds produce in general similar symptoms. Acute cyanide asphyxia is one of the most rapid causes of death with the victim falling dead almost immediately. They are true protoplasmic poisons in that they arrest the activity of all forms of living matter. It is suspected that they may combine with the catalysts of the living cells containing iron or sulphur, thus inhibiting tissue oxidation. They prevent the absorption of oxygen from the blood; the venous blood retains the clear red color of arterial blood. There results a cessation of organic gaseous exchanges with signs of asphyxia, decreased alkalinity of the blood and appearance of lactic acid.

   There is a toxic effect on the central nervous system as shown by signs of paralysis and local action on peripheral nerve endings; etc. Alkaline cyanides in addition are caustic to the skin; itching, papules and vesicules which become infected are frequent. Ulcers may result.

   The complex cyanides such as potassium ferrocyanide, Prussian blue, Trumbull's blue, are considered non-poisonous.

   b. *Hydrocyanic Acid*—The acid and gas exert a gradual but different action; the acid is two to five times as powerful as the gas. Inversely, the gas has a stronger local irritating action on the skin and mucous membranes and sets up a distressing anaesthesia. Cerebral troubles resulting from the acid are more serious than those caused by the gas. In large doses asphyxia is nearly instantaneous, with a cold sweat, dilation of pupils, eyes glassy and staring, loss of consciousness, panting, collapse and death. In acute poisoning, there occurs vertigo, headache, confusion, congestion of head, oppression, palpitation, irritation, dryness and constriction of throat, dyspnea, nausea and vomiting. Later, there follows shivering, sweating, slower pulse, convulsion, abolition of reflexes, involuntary micturition, loss of consciousness, coma with death usually resulting. In less severe cases, recovery from the feeling of constriction in the chest, weakness, unsteady gait, headache, speech difficulties and drowsiness may occur in a few days.

   In subacute poisoning, symptoms are observed such as cough, lassitude, dyspnea, headache, backache, weakness, feeble and rapid pulse, accelerated, irregular and labored breathing, pain in chest and back, vomiting, muscular pain and cramps, trembling, paralysis, disturbances of nervous system. The breath often has the odor of bitter almonds. Color may be pale at first, then red; cyanide rash may be present.

   Chronic poisoning is rare; its existence is denied by some authors. However, symptoms of chronic poisoning have been suggested by some as follows: Headache, vertigo, malaise, feeling of lassitude and weakness; unsteady gait, nausea, vomiting, loss of appetite, disorders of the gastro-intestinal

functions; albuminuria; suppression of tendon reflexes; disorders and irritation of the throat and respiratory system; diminution in cardiac activity, with weak pulse, palpitations and faintings; and diminution in the sensitiveness of the skin.

c. *Sodium Cyanide.* The symptoms are similar to hydrocyanic acid since the poisonous action is produced in the same way but not so powerfully.
There is also marked irritation to skin and nasal mucous membranes. Skin lesions occur as irritating papules or vesicles which are persistent and aggravated by wounds, abrasions, etc. Eczema and acne are frequent. Sites of the lesions are chiefly on the dorsal surface of the fingers and hands, on the wrist, occasionally on the face, ears and back of the neck.

d. *Potassium Cyanide.* See sodium cyanide. Toxic action is similar.

e. *Cyanogen.* The toxicity of cyanogen is similar to and is stated to be about a quarter of that of hydrocyanic acid.

f. *Cyanogen Chloride.* The symptoms are similar to those produced by cyanides with addition of severe irritation to the skin, respiratory system and mucous membranes. Chronic industrial poisoning is stated to result in dizziness, nausea, cough, hoarseness, muscular weakness, staggering, congestion of lungs, loss of weight, prostration, and lachrymation. (All forms of poisoning are severe.) In chronic cases, loss of weight, pulmonary congestion, and cutaneous lesions leads to the supposition that the chlorine is partially responsible.

D. Treatment of Cyanide Poisoning:

1. Sodium Thiosulphate intravenously.
   Sodium Tetrathionate intravenously.
   Methylene Blue intravenously.
2. Artificial respiration.
3. Administration of Pure Oxygen.
4. Lobeline or coramine as stimulants.
5. Usual measures for shock.

E. Sanitary Corps Officer:

1. Cyanide fumes in the plating room
   a. The operation of any solution which has a cathode efficiency of less than 100% gives rise to the evolution of gaseous H which, depending on the type of solution and the current density, either may be involved as fine bubbles which do not disturb the surface of the bath (as with Ag solutions) or else rises with explosive force thus producing a spray of solution (brass and Cr solutions and cyanide pickles and strips).

SELECTED REFERENCES

Agner, Kjell.: Treatment of Hydrogen Cyanide-Poisoned Rabbits with Copper Chloride injections. Naturwissenschaften., vol. 27, p. 31, 1939.

Barcroft, J.: The Toxicity of Atmospheres Containing Hydrocyanic Acid Gas. Abstr. as follows from Jour. Hyg., vol. 31, pp. 1-34, 1931, in Bull. Hyg., vol. 6, pp. 359-360, April 1931.

Bein, G. V.: Hydrocyanic Acid Poisoning. Abstr. as follows from Zentralbl. f. inn. Med., vol. 45, p. 970, Nov. 22, 1924, in Jour. Am. Med. Assn., vol. 84, p. 241, Jan. 17, 1925.

Braddock, William H., M.D., and Tingle, George R.: So-Called Cyanide Rash in gold mine Mill Workers. Jour. Ind. Hyg., vol. 12, no. 7, pp. 259-264, 1930.

Dept. Sci. Ind. Research (Brit.): Hydrogen Cyanide Vapor. Methods for Detection of Toxic Gases in Industry. Leaflet no. 2, p. 9, 1938.

Drinker, Philip: Hydrocyanic Acid Gas Poisoning by absorption through the skin. Jour. Ind. Hyg., vol. 14, no. 1, pp. 1-2, Jan. 1932.

Greene, Robert A.: Report on (the Determination of) Hydrocyanic Acid in Plants. Jour. Assoc. Official Agr. Chem., vol. 21, pp. 354-355, 1938.

Hall, Nathaniel: Cyanide Fumes in the Plating Room. Products Finishing, vol. 3, no. 2, pp. 34-41, 1938.

Hamilton, A.: Industrial Toxicology. Harper and Bros., Publishers, New York, 1934, pp. 152-155.

Henderson, Y., and Haggard, H.: Noxious Cases. The Chemical Catalog Co., Inc., New York, 1927, pp. 110-112.

Ingegno, A. P., and Franco, S.: Cyanide Poisoning. Indust. Med., vol. 6, pp. 573-576, Nov. 1937.

Jour. Am. Med. Assn.: Dangers of Phosphoric and Hydrocyanic Acid Vapors, vol. 79, no. 22, p. 1863, Nov. 25, 1922.

Katz, S. H., and Longfellow, E. S.: Tests for Hydrocyanic Acid Gas in Air. Jour. Ind. Hyg., vol. 5, no. 3, pp. 97-104, July 1923.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., Philadelphia, 1924, p. 651.

Liston, W. G.: Fumigation with Hydrogen Cyanide. Jour. Soc. Chem. In-dust., vol. 44, pp. 367-371, April 10, 1925.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 740-760.

Meyer, Julius., and Hsue-Wen Fan.: Determination of Very Small Quantities of Hydrocyanic Acid. Gasmaske., vol. 11, pp. 17-18. 1939.

Ministry of Health.: Memorandum Issued by the Ministry of Health on the Fumigation of Ships with Hydrogen Cyanide. H. M. Stationery Office, p. 7, 1928. Abstr. as follows in Bull. Hyg., vol. 3, p. 654, Aug. 1928.

Occupation and Health. International Labour Office, Geneva, 1930, pp. 553-560.

Ohio Department of Health, Cyanides, 1940.

Ostmann, B.: Potassium Cyanide and Hydrocyanic Acid Poisoning. Samm. von Vergiftungsf., vol. 9, pp. 33-48, 1938.

Parmenter, D. C., M. D.: Observations on mild Cyanide Poisoning, Report of a Case. Jour. Ind. Hyg., vol. 8, no. 6, pp. 280-282, June 1926.

Paton, W. D.: Cyanide Antidotes. Can. Mining Jour., vol. 59, pp. 135-136, 1938.

Raestrup.: Cutaneous Injuries from Potassium Cyanide. Zentralbl. f. Gewerbehyg., N. S., vol. e, pp. 103-106, April 1926.

Reed, C. I.: Chronic Poisoning from Cyanogen Chloride. Jour. Ind. Hyg., vol. 2, no. 4, pp. 140-143, August 1920.

Rimarski, et al.: Investigation on the Danger of Manufacturing and the safety of transporting and handling Hydrocyanic Acid Containing Cyanogen Chloride, and a New Absorbent. Abstrt. as follows from Jahresbr. Chem. Tech. Reichsanstalt., vol. 8, pp. 71-79, 1930, in Chem. Zentr., vol. 1, pp. 2383-2384, 1931, and Chem. Abstr., vol. 26, p. 4418, 1932.

Rosenbloom, J.: Case of Chronic Hydrocyanic Acid Poisoning. Abstr. as follows from Jour. Lab. and Clin. Med., vol. 8, p. 258, Jan. 1923, Jour. Am. Med. Assn., vol. 95, p. 832, Sept. 13, 1930.

Schultze, W.: Deleterious Action Through the Skin of Poisonous Gases at High Concentrations (Carbon Monoxide, Hydrogen Sulphide, Hydrocyanic Acid, and Aniline). Abstr. as follows from arch. f. Hyg., vol. 98, pp. 70-83, 1927; in Chem. Abstr., vol. 21, p. 3085, Sept. 20, 1927.

Sherrard, G. C.: A Report on the Disposal of Zyklon Following the Fumigation of the Holds of Vessels. U. S. Pub. Health Rep., vol. 42, pp. 3071-3075, Dec. 16, 1927.

Taylor, H.: The Respiratory Movements in Gradual Hydrocyanic Acid Poisoning. Abstr. as follows from Jour. Physiol., vol. 69, pp. 124-134; in Physiol. Abstr., vol. 15, p. 161, June 1930.

Thomas, G. R. S.: Postmortem findings in two cases of Hydrocyanic Acid Gas Poisoning. Abstr. as follows from Lancet., vol. 1, p. 1210, June 16, 1923; in Jour. Am. Med. Assn., vol. 81, p. 508, Aug. 11, 1923.

Ullmann, H.: Cyanide Poisoning from Shoe Cleanser. Abstr. as follows from Deutsch. Med. Wchnschr., vol. 52, p. 451, March 12, 1926: in Jour. Am. Med. Assn., vol. 86, p. 1589, May 15, 1926.

Von Bernewitz, M. W.: The So-Called Cyanide Rash. Jour. Ind. Hyg., vol. 13, no. 3, p. 115, March 1931.

Walton, D. C., and Witherspoon, M. G.: Skin Absorption of Certain Gases. Jour. Pharmacol and Exper. Therap., vol. 26, pp. 315-324, Nov. 1925.

Williams, C. L.: An Unusual Case of Cyanide Poisoning During Fumigation. U. S. Pub. Health Repts., vol. 53, pp. 2094-2095, Nov. 25, 1938.

Williams, C. L.: Calcium Cyanide Dust In Ship Fumigation. U. S. Pub. Health Repts., vol. 51, pp. 139-142, Feb. 7, 1936.

Wirth, W.: Researches on the Treatment of Hydrocyanic Acid Poisoning Caused by Inhalation. Arch. f. Exper. Path. Pharm., vol. 179, pp. 558-602, 1935.

HYDROCARBONS: GASEOUS

Chemical Formula and Synonyms:

(Methane) CH_4, fire damp, marsh gas.
(Ethane) C_2H_6.
(Propane) C_3H_8.
(Butane) C_4H_{10}.
(Ethylene) C_2H_4, bicarburetted hydrogen, elayl, ethene, etherin, heavy carburetted hydrogen, olefiant gas.
(Propylene) C_3H_6, methylethylene, propene.
(Amylene) C_5H_{10}, beta-isoamylene, pental, pentene, trimethylethylene, valerine.
(Acetylene) C_2H_2, ethine.

A. **Principal Properties:**

1. Methane
   a. Tasteless, inflammable and odorless gas; slight garlicky odor; condensable with great difficulty; forms an explosive mixture with air. Sp. gr. 0.554 (referred to air).
   b. Soluble in alcohol and ether; slightly soluble in water and fuming sulfuric acid. Wt. per liter, 0.66 gram.
2. Ethane
a. Colorless gas; sp. gr. (liquid) 0.546 at 88°C., (gas) 1.049 (referred to air).
   b. Soluble in absolute alcohol, slightly soluble in water, Wt. per liter, 1.83 grams.
3. Propane
   a. Colorless gas. Sp. gr. 1.562 (referred to air).
b. Slightly soluble in water; soluble in alcohol and ether. Wt. per liter, 1.83 grams.
4. Butane
   a. Colorless gas. Sp. gr. (liquid) 0.60 at 0°C., (gas) 2.046 (referred to air).
   b. Soluble in water, alcohol, and ether. Wt. per liter, 2.41 grams.
5. Ethylene
   a. Colorless gas with characteristic sweet odor and taste. Inflammable. Sp. gr. 0.975 (referred to air).
   b. Slightly soluble in water, alcohol, and ether. Wt. per liter, 1.17 grams.
6. Propylene
   a. Colorless gas. Sq. gr. (liquid) 0.609 at —47/4°C., (gas) 1.498 (referred to air).
   b. Soluble in water, alcohol, and acetic acid. Wt. per liter, 1.74 grams.
7. Amylene
   a. Colorless, mobile, inflammable liquid, disagreeable odor; readily polymerized. Sp. gr. 0.666.
   b. Soluble in alcohol and ether, very slightly soluble in water. Wt. per liter of vapor, 2.91 grams.
8. Acetylene
   a. Colorless gas; ethereal odor; highly inflammable. Sp. gr. 0.91.
   b. Soluble in acetone, alcohol, and water. Forms explosive mixtures with air over limits of 3 to 82% acetylene. Wt. per liter, 1.08 grams.

B. **Principal Industrial Uses:**

1. Methane
   a. Chief, constituent of most natural gases and of manufactured fuel.
2. Ethane
   a. As a fuel in Natural gas.
3. Propane
   a. Fuel gas; used as a combined solvent and refrigerant for dewaxing and extracting lubricating oils in the petroleum industry; used in the manufacture of ethylene glycol and related products.
4. Butane
   a. Fuel.

5. Ethylene
   a. Organic preparations; production of mustard gas; oxyethylene welding and cutting of metals; medicine (to produce anesthesia), manufacture of synthetic ethyl alcohol.
6. Propylene
   a. Used for the production of isopropyl alcohol, propylene glycol, acetone, isopropyl acetate, isopropyl ether, methyl isobutyl carbinel, propylene chlorohydrin, propylene oxide, proylene dichloride, and methyl isobutyl ketone.
7. Amylene
   a. Local anesthetic.
8. Acetylene
   a. Manufacture of dyes, intermediates, chemicals (acetaldehyde, acetic acid, tetra chlorethane, trichloroethylene, hydrogen, ethylene), explosives, acetylene black, synthetic resins, synthetic rubber, synthetic tannins, enriching illuminating gas; general illuminating purposes; fuel; welding and cutting of metals.

C. Poisoning—Systemic

1. Modes of entry
   a. Industrial poisoning by the Gaseous Hydrocarbons is concerned exclusively with the Inhalation of the gases.
2. General Considerations
   a. The pure gaseous hydrocarbons are physiologically indifferent gases and in themselves have little interest to the industrial toxicologist. However, toxic symptoms may result from exposure to impurities or substances frequently associated with these gases. Such substances may arise through the use of the gases as in combustion or they may be introduced in the production of the gas from impure raw materials.

A classification of the more common gaseous hydrocarbons according to their physiological action is indicated as follows:

Name	*Boiling point*	*Physiological action*
Paraffins		
Methane	—161.4°C.	Simple asphyxiant
Ethane	— 88.0	do
Propane	— 44.5	Simple asphyxiant & anesthetic
Butane	0.6	do
Olefins		
Ethylene	—103.9	do
Propylene	— 48.0	do
Amylene	37—42	do
Acetylene	— 84.0	do

It is emphasized that the higher members of the series have a much stronger anesthetic action than the lower members.

Large numbers of people are exposed to gaseous hydrocarbons and considerable loss of life is caused annually through explosions of these gases. A discussion of the conditions incident to such explosions and their prevention is outside the scope of this bulletin. Detailed information on explosion of gaseous mixtures may be obtained in numerous publications. The U. S. Bureau of Mines has compiled and published much valuable information which may be obtained at a nominal cost.

a. (Paraffin series—methane, ethane, propane, and butane). The action of methane gas is that of a simple asphyxiant. When mixed with air in the proportion of 45% methane the oxygen content is reduced to 11.5% and breathing is somewhat deeper. When a concentration of 70% of methane is reached, life is

endangered. As the colecular weight of the gasseous paraffin hydrocarbons increases there is an increasing anesthetic effect. The presence of carbon monoxide must always be considered as a potential source of danger when these gases are used in combustion processes.

b. (Olefin series—ethylene, propylene, and amylene). Ethylene was first thought to be an irrespirable gas but it is now regarded as relatively harmless, although it has a slight narcotic effect in high concentrations. Layet caused a dog to inhale a mixture of 27 parts of ethylene, 10 parts of oxygen, and 50 parts of air for half an hour without producing any toxic symptoms. The high concentration necessary to produce a marked physiological effect dilutes the air to a level which will not support life. Sixty percent of ethylene or prophylene administered with oxygen has been used to induce surgical anesthesia. The higher members have a stronger anesthetic action than ethylene and propylene, amylene having been employed for surgical anesthesia.

Commercial ethylene has occasionally been contaminated with carbon monoxide and the possibility of carbon monoxide should be kept in mind when ethylene is used in any quantity.

c. Acetylene. Simple cases of poisoning cannot be attributed to pure acetylene although it was thought for a long time that the gas acted like carbon monoxide. Pure acetylene has been used with oxygen as an anesthetic. The patient loses consciousness in one or two minutes and when administration is stopped consciousness quickly returns. The after effects of the anesthesia, such as nausea, vomiting, and headache, are not completely eliminated but disappear quickly.

Crude acetylene prepared from calcium carbide contains 10-14% of impurities including phosphine, carbon disulfide, hydrogen sulfide, silicon hydrode, arsine, antimony, carbon monoxide, hydrogen, nitrogen, and miscellaneous hydrocarbons. The possibility of poisoning by traces, of impurities in acetylene is at once suggested.

It is not surprising that the symptomotology would be obscure. Symptoms of headache, nausea, general malaise, feeling of suffocation, and pain over the liver have been reported. Nervous affection, including excitability, muscular cramp, exaggerated reflexes, and tachycardia, are recorded as sequelae of acetylene poisoning. The extent and character of the symptoms are determined by the nature and amount of impurities in the crude acetylene.

The great danger of acetylene lies in the risk of explosion, a risk which is increased by the presence of certain impurities such as phosphine. The regulations pertaining to the manufacture and use of acetylene have resulted in methods of purification and handling of the gas which is properly enforced greatly diminish the possibility of serious accident.

SELECTED REFERENCES

Bureau of Mines: U. S. Department of Commerce. Limits of Inflammability of Gases and Vapors. Bulletin, no. 279.

Burgess, M. J., and Wheeler, R. V.: The Ignition of Firedamp by the Heat of Impact of Metal Against Rock. Safety in Mines Res. Board Paper, no. 54. H. M. Stationery Office, 1929.

Burgess, M. J., and Wheeler, R. V.: The Limits of Inflammability of Firedamp and Air. Safety in Mines. Res. Board Paper No. 15, H. M. Stationery Office, 1925.

Cotte, E.: Ignition of Firedamp by Portable Incadescent Electric Lamps. Rev. Ind. Minerale, no. 367, pp. 371-373, 1936.

Coward, H. F., and Grice, C. S. W.: Principles of Self-Acting Firedamp Alarms. Tr. Inst. Min. Engin., vol. 81, p. 350, June 1931.

Coward, H. F., and Wheeler, R. V.: The Ignition of Firedamp. Safety in Mines Res. Board, Paper, no. 53, H. M. Stationery Office, 1929.

Hamilton, A.: Industrial Toxicology Harper and Brothers, New York, 1934, pp. 90, 233.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 89, 96-103, 149-155.

Heyer: Firedamp and Its Detection. Ztschr. f. d. ges. Schiess-u Sprengstoffw., vol. 24, pp. 164-169, 1929.

McNally, W.: Toxicology. Industrial Medicine. Publishers, Chicago, 1937, pp. 379-385.

Mawich: Fatal Poisoning from Welding with Acetylene (Dissous Gas) Arbeitsschutz, pp. 43-44, 1938.

Maxwell, G. B., and Wheeler, R. V.: Fire Damp Explosions within Closed Vessels. The Effects of Turbulence. Safety in Mines Res. Board, Paper, no. 10. H. M. Stationery Office, 1925.

Miller, Alex. U., and Thomas, Edward: Hazards of Methane from Well Water in Some Sections of Illinois. U. S. Bur. Mines, Circ., no. 7033, 5, pp. 1938.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 38-44, 839.

Ohio Department of Health, Gaseous Hydrocarbons, 1940.

Perepelitsa, V. K.: Apparatus for Detection of Methane. Ugol., no. 1, pp. 61-64, 1939.

Selivanoff, E.: An Improved Method for Analyzing the Air of Mines by Determination of Methane and Carbon Dioxide. Gigiena Truda, no. 3, pp. 34-37.

Smith, H. I., and Hamon, R. J.: Methane Accumulations from Interrupted Ventilation. U. S. Bur. of Mines, Tech. Paper 190.

FLUORINE: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Fluorine) F_2.
(Hydrofluoric Acid) H_2F_2, fluohydric acid, hydrogen fluoride.
(Sodium Fluoride) NaF, fluorol.
(Silicon Fluoride) SiF_4, silicontetrafluoride.
(Cryolite) Na_3AlF_6, cryolith, greenland spar, ice stone, kryolith.
(Ammonium bifluoride) NH_4HF_2.

A. **Principal Properties:**

1. Fluorine
   a. Colorless gas; corrosive; poisonous; Sp. gr. (liquid) 1.11, (gas) 1.31 (A); m.p. —223°C.; b.p. —187°C. Decomposes in water.
2. Hydrofluoric Acid
   a. Clear, colorless, fuming, mobile, corrosive liquid. Produces terrible sores when allowed to touch the skin. Sp. gr. 0.988; m.p. (anhydrous liquid) —83°C.; b.p. (anhydrous liquid) 19.44°C. Soluble in water. Wt. per liter of gas, 0.83 gr.
3. Sodium Fluoride
   a. Clear, lustrous crystals or white powder; poisonous. Sp. gr. 2.79; m.p. 992°C. Soluble in water; slightly soluble in alcohol.
4. Silicon Fluoride
   a. Colorless gas; suffocating odor; fumes strongly in air. Absorbed readily in large quantities by water with partial decomposition. Sp. gr. 3.57 (A), m.p. —77°C., b.p. —65°C. at 1810 mm.
5. Cryolite
   a. A natural fluoride of sodium and aluminum. Usually colorless to snow white, but sometimes reddish or brownish or even black. Vitreous, greasy, moist-looking or pearly luster. Sp. gr. 2.9 to 3; hardness 2.5.
6. Ammonium bifluoride
   a. White crystals. Sp. gr. 1.211. Soluble in cold water; decomposes in hot water.

B. **Principal Industrial Uses:**

1. Fluorine
   a. Organic synthesis; fluorine compounds.
2. Hydrofluoric Acid
   a. Chemicals (fluorides, electrolytic manufacture of chlorates and persulfates, hydrogen peroxide from peroxide of sodium; analytical reagent; ceramics (to increase porosity); breweries and distilleries (antiseptic, retarding injurious fermentation); frosted glassware; etching glass; reagent in manufacture of filter paper; purification of beet sugar; yeast manufacture; manufacture of chemical and physical apparatus, for etching divisions on thermometer stems, etc.; cleaning copper and brass; removal of sand particles in metallic castings; graphite purification.
3. Sodium Fluoride
   a. Antiseptic and antifermentative in alcohol distilleries, etc., food preservative; roach and rat poison; medicine; flux; enamels.
4. Silicon Fluoride
   a. Manufacture of fluosilicic acid; chemical analysis; nuisance by-product in fertilizer manufacture.
5. Cryolite
   a. Chemicals (sodium salts); aluminum manufacture (flux); glass (opacity); manufacture of vitreous enamels.
6. Ammonium Bifluoride
   a. Ceramics; chemical reagent; etching glass (White acid); sterilizer for brewery, dairy, and other equipment.

C. Poisoning—Systemic:

1. Modes of Entry
   - *a.* Inhalation.
   - *b.* Ingestion.
2. Symptoms
   - *a.* Fluorine: This gas in the elementary state is rare and has little industrial significance. It is extremely active and even more corrosive than hydrofluoric acid. In the presence of moisture, fluorine gas is quickly converted into hydrofluoric acid and ozone (see hydrofluoric acid).
   - *b.* Hydrofluoric Acid: It is intensely irritating and caustic and when inhaled may result in coryza, bronchial catarrh with spasmodic coughing, a sense of constricted breathing and pulmonary edema. It causes irritation and ulceration of mucous membranes; also may cause lachrymation and salivation. The damage is generally limited to severe dermatitis, often with vesicles and necrotic ulcers which become indurated and difficult to heal. May cause painful ulcers of the cuticle, erosion and formation of vesicles, suppuration under the fingernails.
   - *c.* Sodium Fluoride: Chronic poisoning causes symptoms like alkaline compounds; if ingested is extremely caustic. It is a general protoplasmic poison and has a strong local irritant action. Absorption of small amounts (fractions of a gram) of the salts can result in symptoms of nausea and vomiting, gastric pain, salivation, pruritis and diarrhea. Larger amounts (over one gram) of the salts may cause vomiting, cramps, fibrilliary tremors, rigidity followed by muscular paralysis, acceleration followed by paralysis of respiration and paralysis of the central nervous system. Ingestion of larger amounts of salts give rise to acute poisoning with rapid fatal termination.
   - *d.* Cryolite: Chronic fluorosis from the inhalation of this dust has been reported in Europe with mottling and degenerative changes in the teeth and osteosclerosis with ligament calcification. There is also loss of weight, dyspnea on exertion, loss of appetite and vomiting, with some anemia. These may disappear when exposure ceases.
   - *e.* Ammonium Bifluoride: Chronic poisoning may produce symptoms of vomiting, cramps, tremors, muscular spasticity, difficult respirations, and decrease of blood calcium.

SELECTED REFERENCES

Brailsford, J. F.: Radiological Demonstration of Pathological Changes Induced by Certain Industrial Processes. Brit. Jour. Radiol., vol. 11, pp. 393-400, June 1938.

Cannava, A.: Fluorine Poisoning II. Arch. Ital. Sci. Farmacol., vol. 6, p. 456-468, 1937; Chimie and Industrie., vol. 40, pp. 647-675.

Cirla, P.: The Phosphorus Content of Blood and of Bone in Various Types of Experimental Poisoning; Lead, Mercury, Chromium, Phosphorus, Strontium and Fluorine. Med. del Lav., vol 28, pp. 44-54, 1937.

Gudjonsson, Sk. V., M. D.: A Study of 78 Workers Exposed to Inhalation of Cryolite Dust. (From the Department of Occupational Hygiene, Industry, Inspectiorate, Copenhagen.)

Hamilton, A.: Industrial Toxicology. Harper and Bros., Publishers, New York, 1934, pp. 16-17.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, p. 128.

Kempf, C. A., Greenwood, D. A., and Nelson, V. E.: Studies Relating to Toxicity of Fluorine Compounds. Jour. Lab. and Clin. Med., vol. 22, pp. 1133-1137, Aug. 1937.

Kober, G., and Hyhurst, E.: Industrial Health. P. Blakiston's Son and Co., Publishers, Philadelphia, 1924, p. 653.

Kockel, and Zimmerman.: Intoxication with Fluorine Compounds. Abst. as follows from Munchen. Med. Wchnschr., vol. 67, pp. 777-779, 1920.

Lain, Everett S.: Fluoride Toxicity: A Public Health Problem. Southwest Water Works Jour., vol. 20, no. 11, pp. 23-24, 1939.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago. 1937, pp. 91-108.

Marx, I.: The Effect of Sodium Fluoride on Experimental Osteodistrophy and Atrophy from Inactivity. Bruns Beitrage z. Klin. Chir., vol. 168, pp. 261-266, 1938.

Occupation and Health, International Labour Office, Geneva, 1930, pp. 789-793.

Ohio Department of Health, Fluorine and Its Compounds, 1940.

Roholm, K.: Fluorine Compounds. Suppl. to Occupation and Health, International Labour Office, p. 7, Sept. 1938.

Roholm, K.: Fluorine Poisoning, A New Disease. Klin. Wchnschr., vol. 15, pp. 1425-1431, 1936.

Roholm, K.: Fluorine Poisoning In Cryolite Workers. Arch. Gewerbepath. f. Gewerbehyg., vol. 7, pp. 255-277, 1936.

Rost, E.: Toxicology of Fluoride. Arch. f. Gewerbepath. u. Gewerbehyg., vol. 8, pp. 256-265, 1937.

Scheuermann, H.: The Effect of Hydrofluoric Acid on the Skin. Dermatol. Wchnschr., vol. 104, pp. 661-667, 1937.

Smith, H. F., and Smith, H. F., Jr.: Relative Toxicity of Some Fluorine and Arsenical Insecticides. Indust. and Engin. Chem., vol. 24, p. 229, 1932.

Velu, H.: Report on Fluorine Poisoning and Diseases of the Teeth in Sheep. Compt. Rend. Soc. de Biol., vol. 127, pp. 854-855, 1938.

Weber, H. H., and Engelhardt, W. E.: An Apparatus for Producing Low Dust Concentrations of Constant Composition and a Method for the Microgravimetric Determination of Dust. The use of the Method in Studying Dust from the Production of Beryllium. Zentralbl. f. Gewerbehyg. N. S., vol. 10, pp. 41-47, Feb., Mar., 1933.

Wolff, W. A., and Kerr, E. G.: The Composition of Human Bone in Chronic Fluoride Poisoning. Am. Jour. Med. Sci., vol. 71, p. 185, 1938.

Ziener, Theo.: Safety Measures in the Use of Hydrofluoric Acid. Glass., vol. 15, pp. 240-242; Sprechsaal., vol. 71, p. 185, 1938.

HYDROGEN SULFIDE

Hydrogen Sulfide is an irritant and toxic gas. It causes irritation of the entire respiratory system and of the conjunctiva of the eye, and in high concentrations it may produce respiratory paralysis and neurological changes.

A. Principal Properties:

1. Hydrogen Sulfide, H_2S, is a colorless gas having an offensive odor, as of rotten eggs at low concentration, and a sweetish odor at high concentration.
2. Combustible in concentration of between 4.4 and 44.5 per cent in air.
3. Molecular weight 34.08.
4. Specific gravity 1.192.

B. Principal Industrial Uses:

1. Chemical industry as in the manufacture of carbon disulfide, of sulphur dyes, and of soda according to the Le Blanc process; in the manufacture of glue; in washings from sugar beets and in sewer gases.
2. Rubber and rayon industry.

C. Poisoning—Systemic:

1. Modes of entry.
   a. Absorption through the lungs.
   b. Absorption through the skin is doubtful.
2. Symptoms.
   a. Acute; with high concentration the victim may suddenly collapse and die as a result of respiratory paralysis. With less concentration there may be a progressive depression of respiratory center. With still less serious exposure the irritant action of hydrogen sulphide may be predominant, resulting in irritation of the mucous membrane of the eye and of the respiratory tract causing possibly a pulmonary edema and bronchial pneumonia.
   b. Chronic exposure; continued exposure to low concentration may cause fatigue, headache, especially in the temples, and such nervous conditions as irritability and sleeplessness.
   c. The effects of hydrogen sulfide on the gastro-intestinal tract, such as loss of appetite, loss of weight, nausea and vomiting, have been associated with exposure to hydrogen sulfide.
   d. Late effects of hydrogen sulfide poisoning may result in inflammatory process of the respiratory tract or in circulatory disturbances characterized by bradycardia and temporary weakening of the cardiac muscle, peripheral neuritis, lymphocytosis and gastro-intestinal disturbances.

D. Mechanism of Action of Hydrogen Sulfide:

1. It is claimed that hydrogen sulfide interferes mainly with the oxygen metabolism of the cell by affecting the iron-containing ferment and that the primary stimulation of the respiration is not due to irritation but is a sign of beginning anoxia.

E. Toxic Concentrations:

1. The maximal permissible concentration of hydrogen sulfide in air is accepted at present as 20 parts per million by volume, corresponding to 0.028 mgm. per liter of air at 25° C and 760 mm. Hg. for exposures not exceeding a total of eight (8) hours daily.

F. Treatment:

1. Transfer of patient to fresh air as quickly as possible.
2. Artificial respiration and oxygen.
3. Prevent chilling.
4. Irritative symptoms of the respiratory tract treated symptomatically.
5. The conjunctivitis may be helped by instillation of a few drops of olive oil.

G. Sanitary Corps Officer:

1. Strict attention in all operations where hydrogen sulfide may contaminate the air, the concentration of 20 parts per million be maintained by proper exhaust ventilation, preferably at the site of the formation of the gas.

H. Instructions to Operators:

1. Whenever rooms or enclosures have to be entered, which may contain hydrogen sulfide, this should be done only with open air masks, safety belts, and under the supervision of a crew familiar with the potential dangers of such exposure, and with the proper first aid measures.

SELECTED REFERENCES

Barrat, P., and Seigner, A.: A Little Known Industrial Disease: Gas Poisoning from Certain Under-Water Materials Encountered by Workers in Diving Bells. Ann. d'oculist., vol. 98, pp. 513-527, 1936.

Clark, H. P.: Sulfuretted Hydrogen Poisoning: with Special References to Eye Symptoms—"Pink Eye." Med. Jour. Australia, vol. 1, pp. 439-440, May 3, 1924.

Edington, J. W.: Sulfuretted Hydrogen Produced by Bacteria in a Lead Mine. Jour. Hyg., vol. 38, pp. 683-687, Nov. 1938.

Eggert. J.: Poisonous Gases in a Viscose Rayon Plant and Their Removal, Chem.—Ztg., vol. 52. pp. 505-506, 1928.

Fowler, H. C.: Poisoning from Hydrogen Sulfide. Oil and Gas Jour., vol. 170, no. 36, pp. 173-174, 1928.

Haggard, Howard W., M. D.: The Toxicology of Hydrogen Sulfide. Jour. Ind. Hyg., vol. 7, no. 3, pp. 113-121, March 1925.

Hamilton, A.: Industrial Toxicology. Harper and Bros., Publishers, New York, 1934, pp. 147-152.

Hartmann, K.: Surface and Deep Corneal Inflammation (Disc Shaped) Caused by Hydrogen Sulfide in Caisson Workers on the North Sea Coast. Klin. Monatsbl. f. Augenheilk., vol. 99, pp. 456-468, 1937.

Hellfors, A.: Fatal case of Angina Pectoris After Breathing Hydrogen Sulfide Gases. Munch. Med. Wchnschr., vol. 85, p. 1149, 1938.

Israelson, Z. I., and Voltaire, S. V.: Acute Industrial Poisoning in Tanneries. Med. du. Trav., vol. 6, pp. 263-286, Sept. 1934.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., Philadelphia, 1934, pp. 365-661.

Krahnstover, M.: Injury to the Eyes from Hydrogen Sulfide. Zentralbl. f. Gewerbehyg., N. S., vol. 5, pp. 245-247, Aug. 1928.

Lange, R.: Fatal Hydrogen Sulfide Poisoning in the Settling Plant of a Paper Mill. Samm. v. Vergiftungs., vol. 6, pp. 233-236, 1935.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 390-400.

Malov, G. A., Nikolaev, M. G., and Rudenko, E. I.: Protective Substances in Hydrogen Sulfide Poisoning. Arch. Intern. Pharmacodynamie., vol. 56., pp. 232-237, 1937.

National Institute of Health, Division of Industrial Hygiene, Hydrogen Sulfide, Its Toxicity and Potential Dangers, 1941.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 979-987.

Ohio Department of Health, Hydrogen Sulfide, 1940.

Pennsylvania Department Labor and Industry, Occupational Disease Prevention Division; Survey of Carbon Disulfide and Hydrogen Sulfide Hazards in the Viscose Rayon Industry. Bull. no. 46, 69 pp. Harrisburg, Pa., 1938.

Pomilio, U.: Industrial Hygiene and Its Relation to the Manufacture of Artificial Silk in Italy. Gior. Chim. Indust. Applicata., vol. 10, pp. 7-11, 1928.

Reed, J. B.: An instrument for Indicating and Recording Traces of Hydrogen Sulfide in Coal Gas. Jour. Soc. Ind., vol. 57, pp. 43-44, 1938.

Rodenacker, G.: Eye Irritations of Spinners in Artificial Silk Factories. A Contribution to Hydrogen Sulfide Poisoning. Zentralbl. f. Gewerbehyg., N. S., vol. 3, pp. 20-23, Jan. 1926.

Rodenacker, G.: Hydrogen Sulfide—The Mechanism of Its Toxicity. Zentralbl. f. Gewerbehyg., N. S., vol. 4, pp. 372-377, Oct. 1927, pp. 405-409, Nov. 1927.

Schmitt, F. O., and Beck, L. V.: The Effect of Carbon Monoxide and of Hydrogen Sulfide on Nerve Irritability. Biol. Bull., vol. 59, pp. 269-274, Dec. 1930.

Segal, B.: Sulfuretted Hydrogen as a Mining Hazard. Jour. Chem. Min. Met. Soc. So. Afr., vol. 36, pp. 366-377, June 1936.

Sjogren, H.: Contribution to Our Knowledge of the Ocular Changes Induced by Hydrogen Sulfide. Acta Ophth., vol. 17, p. 166, 1939.

Stocker, F.: Practical Observations on the Pathology and Treatment of the Action of Sulfuretted Hydrogen Gas on the Eyes. Med. Klin., vol. 27, pp. 167-168, Jan. 27, 1931.

Yant, W. P., and Fowler, H. C.: Hydrogen Sulfide Poisoning in the Texas Panhandle, Big Lake, Texas, and McCamey, Texas, Oil Fields. U. S. Bur. Mines, Rep. Investigations, Serial No. 2776, p. 20, Oct. 1926.

LEAD

A. **Principal Properties:**

Heavy, malleable, ductile, gray, soft metal.

B. **Principal Industrial Uses:**

1. Paint and pigments.
2. Storage battery manufacture.
3. Type metal and other alloys.
4. Anti-knock gasoline (tetraethyl lead).
5. Insecticide.
6. Rubber Industry.

C. **Lead Poisoning:**

1. Modes of entry.
   - *a.* Inhalation of dust and fumes.
   - *b.* Ingestion of lead compounds.
   - *c.* Skin absorption—e.g., tetraethyl lead.
2. Symptoms.

Suggestive Evidence of Lead Absorption.	*Suggestive Evidence of Incipient Intoxication.*	*Suggestive Evidence of Plumbism.*
a. General Appearance.		
Restive, moody, easily excited, "flustered."	Pallor.	Anemia.
Lead line.	Lead Line.	Lead Line.
	Jaundice.	Jaundice.
		Emaciation.
		"Premature Aging."
b. Digestive System.		
Persistent Metallic Taste.	Metallic Taste.	Metallic Taste.
Slight loss of appetite.	Definite loss of Appetite.	Increased loss of appetite.
Slight constipation.	Coated Tongue.	Nausea and Vomiting.
	Slight abdominal colic.	Coated Tongue.
	Constipation.	Marked abdominal colic.
		Rigid Abdomen.
		Marked constipation.
		Blood in the stool.
c. Nervous System.		
Irritable.	Slight Headache.	Increased Headache.
Uncooperative.	Insomnia.	Increased Insomnia.
	Slight Dizziness.	Increased Dizziness (Ataxia)
	Palpitation.	Confusion.
	Increased Irritability.	Marked reflex changes.
	Increased reflexes.	Tremor.
		Fibrillary Twitching.
		Neuritis.
		Visual disturbances.
		Encephalitis (Hallucinations, convulsions, coma).
		Paralysis.
d. Miscellaneous changes.		
None.	Muscle soreness.	General Weakness.
	Easily fatigued.	Joint Pains.
	Hypotension.	Hypertension.

Suggestive Evidence of Lead Absorption.	*Suggestive Evidence of Incipient Intoxication.*	*Suggestive Evidence of Plumbism.*
e. Urine Examination.		
Positive test for Lead.	Positive test for Lead. Trace of Albumin. Few Granular Casts.	Positive test for Lead. Increase in Albumin and casts. Hematoporphyrin present. Hematuria.
f. Blood Changes.		
Polycythemia. Polychromatophilia. Increased Platelets. Percentage of reticulocytes about doubled.	Normal red blood cell count and normal hemoglobin. Further increase in reticulocytes. From 50 to 100 stippled cells per hundred thousand erythrocytes.	Decrease in hemoglobin. Decrease in total number of red blood cells. Increase in all form of basophilic cells. Increase in percentage of mononuclears. Anisocytosis and poikilocytes. Nucleated red cells present in the peripheral circulation. Decreased platelets.

3. Toxic Concentration.
   1.5 mg. Lead per 10 cubic meters is recognized safe limit.

D. Sanitary Corps Officer:

1. Frequent inspection of exhaust system and ventilation.
2. Determination of extent of atmospheric pollution by air analysis.
3. Proper wash facilities should be available with insistence that employees observe rules regarding washing before meals to avoid ingestion of lead.

E. Medical Control:

1. Preemployment Examination
   a. An occupational history as to prior exposure.
   b. Those who have degenerative cardiovascular disease, diabetes, endocrine disturbances, syphilis or tuberculosis, should not be employed as lead workers.
   c. Complete physical examination, x-ray of chest, Kahn test, urinalysis, total W.B.C., differential, stippled cells per 50 fields.
2. Periodic Examination
   a. When engineering control measures and air analysis indicate that no exposure hazard exists, re-examinations every six months. Included in recheck at six months besides the blood analysis, a quantitative estimation of lead in the urine should be made.
   b. When exposure to lead exists, re-examination every two weeks to every three months, depending on the intensity of exposure. A gradual rise of stippled cells in 50 fields from day to day is evidence of an impending acute Plumbism that may be prevented. The results of the periodic recheck must be analyzed frequently so that proper recommendations may be made for further precautions against exposure.
3. Treatment
   a. Removal from exposure.
   b. Calcium therapy for the acute manifestations.
   c. Deleading program is at times a dangerous procedure. With proper control measures of exposures, the absorbed and stored lead returns to its normal balance after a period of time.
4. The Basophilic Aggregation Test for Lead Absorption and Lead Poisoning
   a. The need for a simple test for lead absorption and incipient lead poisoning has long been recognized. To make this test acceptable, it must fulfill several requirements. It must be a relatively simple procedure which every practitioner or technician can do in a few minutes. It must be as accurate as any accepted physiological determination and it must be approved by the test of time.

b. When the individual has absorbed lead in amounts greater than traces, one of the first body reactions is the formation of a larger than normal number of immature red cells and their appearance in the peripheral circulation. These immature blood cells may be distinguished from older red blood cells by several peculiar characteristics, the chief of which is the presence of basophilic material.

c. Attention must be called here to the difference in the terms basophilic aggregation, basophilia, polychromatophilia, reticulocytes, and preformed stippled cells which exhibit punctuate basophilia. Basophilic aggregation is understood to mean a clumping of basophilic material into intracellular reticular forms so that a totality of all cells containing basophilic material may be determined. In the basophilic aggregation, the basophilic substances within the red cells may show fragmentation, combined reticulation and granulation, bands and wreaths at the periphery, coarse and fine stippling, balled or clumped basophilic material near the center of the cell, distended reticulum, and other forms. Hence, the number of basophilic aggregation cells seen in a smear stained by the technique subsequently described will be the total number of punctate stippled cells, reticulocytes, and all other cells which exhibit basophilic material.

d. Preparation of the slides.

It is important that slides of good quality be used and it is preferable to discard them (for basophilic aggregation tests) after they have been used once. These slides must be grease free as any great variance in the thickness and evenness of the smear will give erroneous results. The following procedure is recommended for cleaning slides:

(*a*) Place the slides in cleaning solution, consisting of concentrated sulphuric acid to which has been added a few crystals of potassium dichromate. It is recommended that the slides be allowed to remain in this solution for 3-4 days. (If time is not available the slides may be rinsed thoroughly in the warm cleaning solution.)

(*b*) Rinse the slides thoroughly in tap water which is preferably hot.

(*c*) Transfer the slides to distilled water and then to 80% ethyl alcohol.

(*d*) Polish the slide with cheese cloth.

(*e*) Flame thoroughly and cool.

Note: The face and ends of the slide should not come into contact with grease from the fingers.

e. Preparation of the Blood Smear.

A thin, even smear covering at least half of the length of the slide is desirable. Thinness should be such that 150 to 200 cells may be seen under the oil immersion objective. The following technique is to be employed:

(*a*) Obtain blood from finger or lobe of ear after cleaning part with alcohol and drying.

(*b*) Do not squeeze the blood out of the puncture.

(*c*) Do not use the first drop of blood.

(*d*) In order to obtain a thin smear, avoid taking too large a drop of blood.

(*e*) The drawing out or "pushing" of the blood droplet with the beveled edge of a second slide may be employed according to the technique to which one is accustomed.

f. Drying of the Blood Smear.

The proper drying of the blood smear is of utmost importance. According to Dr. C. P. McCord who first described this diagnostic test, "If the smear is permitted to become extensively dry, that is, longer than twelve hours, some of the basophilic containing cells will not lead themselves to aggregation of their basophilic material. On the other hand, insufficient drying facilitates removal of the cell during the staining period. Ordinarily, the optimum times lies between one to three hours." M. C. Hyler, however, has found that smears may be stained as late as sixty hours and accurate results obtained. It has been the experience of this Division that the optimum time for staining is from six to twelve hours.

g. Fixation of one-half of the Smear.

After the smear has dried, one-half of the slide, on a longitudinal basis, is fixed. A faulty technique at this period may introduce sources of errors. Precaution must be taken that vapors from the alcohol used for fixing the slide do not partially fix the other half.

Several methods of fixing half of the slide may be employed. Dr. R. R. Jones of the U. S. Public Health Service prefers covering the longitudinal half of the slide with a strip of filter paper which is cautiously moistened with an absolute methyl alcohol (acetone free). An excess of alcohol is easily added and tends to run on the portion of the slide which is not to be fixed. Another method, the dipping of the longitudinal third of the slide was devised by M. C. Hyler. This is particularly

useful when large numbers of slides are to be stained. A shallow utensil such as a petrie dish is filled with alcohol to such depth that one-third of the slide is submerged by dipping to the bottom of the utensil.

After instantaneous dipping the slide is allowed to dry horizontally on muslin or gauze in such a fashion as not to permit any drainage on that portion of the slide for which fixation is wanted. This method is employed when large number of slides are to be stained. A third method of fixing is a modification of the Jones' method and consists of moistening the filter paper before it is placed on the slides, thus decreasing the danger of excess alcohol flowing to the unfixed portion. As soon as the filter paper is moistened, it is placed on the slide, covering a longitudinal half. The moistened filter paper is allowed to remain on the slide until it is dry.

b. Staining the Smear.

The slide is submerged for approximately 10 minutes in either Sussman-Weindel or modified Manson stain. The time is not of great importance as a reliable stain may be obtained in as short a time as two minutes, and on the other hand, it is impossible to over-stain. The staining of the unfixed portion of the slide is in effect a laking process which removes the hemoglobin and clumps the basophilic material rendering it more visible under the microscope.

The formula is as follows:

Toluidine blue	0.5 gm.
Borax	0.05 gm.
Methylene blue (Loeffler's)	5 cc.
Distilled water	100 cc.

The borax is added to the water which is heated. The toluidine blue is added and allowed to stand for a few minutes. Occasional stirring may hasten the solution. The methylene blue is then added. The solution is then filtered through a single No. 30 filter paper.

The Basophilic Aggregation Test.

We have used the Sussmann-Weindel stain extensively, but have encountered difficulties in obtaining uniform toluidine blue. The modified Manson stain as used by the Ohio Department of Health is prepared as follows:

Sodium borate C. P.	1.0 gm.
Methylene blue powder (Loeffler's)	2.0 gm.
Distilled water	100.0 cc.

The water is brought to boiling and the sodium borate is added. After allowing the water to cool to room temperature, the methylene blue is added with vigorous stirring. The solution is then filtered. This stain is stable for at least two weeks. If it is kept in Coplin jars, the surface should be gently skimmed with a piece of filter paper before using.

Dr. Vela-Gonzales states that he has experienced considerable difficulty with the methylene blue and eliminates it entirely by using the following formula. It should be filtered before using.

Toluidine blue	2 gm.
Borax	2 gm.
Distilled water	100 cc.

i. Basophilic Aggregation Counting

In counting the cells, an oil immersion objective and a 10 x ocular provided with a Whipple grid are used. The Whipple grid may be secured from any medical supply house at a nominal price. The outlines of the grid determine the microscopic field. The slide is first examined under low power to discover a uniform area in which the cells are well separated and of even distribution. Final examination is made with the oil immersion objective without the use of a cover slip. It will be noted that all the cells on the fixed portion of the slide are deeply stained, while on the laked portion of the slide, the normal erythrocytes appear as very faint shadows with indistinct cell outlines. The basophilic material in the red cells in the laked portion stands out sharply as coarse granules or as a reticulated network accomodated to the field before the cells can be seen.

Determination of the percentage of basophilic aggregation cells is done in the following manner: A field is selected in an even portion of the slide on the fixed side. The total number of cells outlined by the grid in this field are counted. A mechanical counter facilitates the counting. The slide is then moved directly opposite to the unfixed laked half of the smear and the cells containing basophilic aggregation are counted in four contiguous fields. The slide is then moved opposite to the fixed side again and another field is counted. The procedure described in the preceding sentences is repeated until five fields have been counted on the fixed side and twenty fields on the laked side (see diagrams on the following page).

The results are expressed in percentage. Thus, if a total of 60 cells with basophilic aggregation was found in the 20 fields on the laked portion and 600 cells in the 5 fields on the fixed portion, the results would be 60 divided by 4 equal 15 divided by 600 or 2.5%. Thus, 2.5% of the total erythrocytes are basophilic aggregation cells.

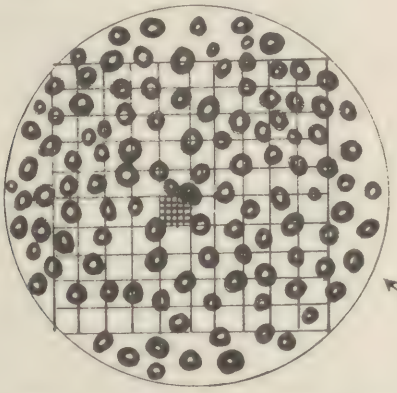
The Basophilic Aggregation Test.

It must be borne in mind that all the cells exhibiting basophilic aggregation may not contain equal amounts of basophilic material and hence will not stain in equal intensity. Therefore, any cells showing any variation in color from the normal must be carefully examined for basophilic aggregation by focusing with the fine adjustment. The basophilic cells should not be counted in the laked portion in the immediate vicinity of the line of separation from the fixed portion of the slide as alcohol may have flowed and partially fixed some cells in this region.

Interpretation of Results.

In interpreting the results of the Basophilic Aggregation test, as with other physiological tests, it is important to consider the medical and industrial history of the patient. In normal adults, cells containing basophilic aggregates rarely exceed 1% of the total number of erythrocytes, but in lead exposed individuals the percentages ordinarily lie above this normal maximum. In the absence of other pathology any finding in lead exposed workers of percentages in excess of 1½% and particularly in excess of 2% suggests lead absorption and the possibility of approaching clinical lead poisoning. In chronic lead poisoning this test usually is not, but may be, positive. As lead poisoning progresses to extended chronicity, the reliability of the procedure diminishes.

In persons exposed both to lead and to other substances such as benxol, toluol, xylol, etc., abnormal percentages of basophilic containing cells may not be caused by the lead but by those other substances or by both. Arsenic may also cause an increase in these cells. Anemias and other types of diseases involving the red blood cells may cause the normal ranges to be exceeded and also occasionally certain infectious diseases.

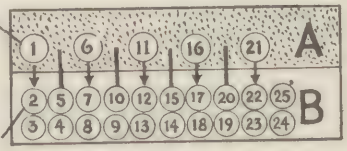


Section of fixed unlaked portion of smear. Determine total erythrocyte count and cellular abnormality. 10X Ocular and oil immersion. Straight lines are from Whipple Grid.

The BASOPHILIC AGGREGATION TEST

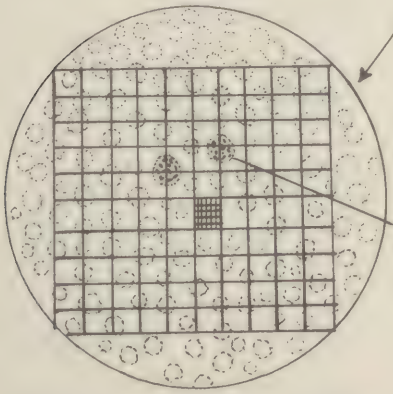
Figure 1

Semi-schematic Drawings



A. Fixed side (alcohol treated). Determine total cells per field.

B. Laked side. Determine basophilic cells per field.



Laked portion of blood smear showing two basophilic aggregation and faint outlines of non-basophilic containing erythrocytes. 10X Ocular and oil immersion.

Semi-schematic drawing of a typical basophilic aggregation containing cell. This is not a preformed punctate stippled cell.

F. **Suggested Examination for Lead Workers.**

1. Name, Address, Age, Race, Sex.

2. History of Previous Exposure to Lead:

   a. Hours per week
   b. Hours per day

3. History and Physical:

   a. Weakness
   b. Drowsiness
   c. Insomnia
   d. Headache
   e. Vertigo

4. Bowels—Number Times Without Cathartics:

   a. Constipation
   b. Diarrhea
   c. Metallic Taste
   d. Anorexia
   e. Nausea
   f. Vomiting
   g. Abdominal pain
      (1) Where
      (2) When
   h. Weight, Average
   i. Muscle Tremor
   j. Muscle Tingling
   k. Muscle Cramps
   l. Joint Pains
   m. Acute Illness

5. Physical Examination Periodic:

   a. Pulse—Temperature
   b. Pallor
   c. Lead Line—Number of Teeth
   d. Right Wrist Extension
   e. Left Wrist Extension
   f. Blood Pressure
   g. Heart
   h. Abdomen
   i. Reflexes

6. Laboratory Examinations:

   a. Stippling—Number in 50 Fields
   b. Hemoglobin
   c. Red Blood Count
   d. White Blood Count
   e. Urine
   f. Urinary Lead

7. Remarks:

McCord, Carey P.; Minster, Dorothy K., and Rehm, Mathilde: "The Basophilic Aggregation Test in Lead Poisoning," J. A. M. A., 82:1759 (May 31), 1942.

McCord, Carey P.; Holden, F. R., and Johnson, Jan: "The Basophilic Aggregation Test for Lead Absorption and Lead Poisoning—Ten Years After Its First Use," *Indust. Med.*, 4:180, April, 1935.

McCord, Carey P.; Holden, F. R., and Johnson, Jan: "Basophilic Aggregation Test in the Lead Poisoning Epidemic of 1934-1935." *Am. J. Pub. Health*, 25:1089, Oct. 1935.

McCord, Carey P.; Walworth, Herbert T.; Johnson, Jan, and Fisher, Philip: "The Atmospheric Content of Lead and Its Correlation with Basophilic Aggregation Tests in Exposed Storage Battery Workers," *Indust. Med.*, 6:357, June 1937.

Ohio Department of Health—Basophilic Aggregation Test, 1940.

Jones, R. R.: "Estimation of Basophilic Cells (Reticulocytes) in Blood by Examination of Ordinary Blood Film," Public Health Reports, 48:1011, Aug. 1933.

Hyler, Muril C., and Bradley, William R.: The Basophilic Aggregation Test," *Indust. Med.*, 4:184, April 1938.

Aliavdin, N., and Peregood, E.: Lead Content in the Duodenal Juice in Cases of Saturnism. Preliminary Repor. Jour. Ind. Hyg., vol. 18, no. 3, p. 139, March 1936.

Aub, Joseph C., Fairhall, Lawrence T., Minot, A. S., and Reznikoff, Paul: Lead Poisoning. Jour. Ind. Hyg., vol. 7, no. 11, pp. 531-537, Nov. 1925.

Aub, J. C., Minot, A. S., Fairhall, L. T., and Reznikoff, P.: Recent Investigations of Absorption and Excretion of Lead in the Organism. Jour. Am. Med. Assn., vol. 83, pp. 588-591, Aug. 23, 1924.

Barnes, E. C.: Possibilities of Control of Lead Exposure by Examining Less Than 24 Hour Urine Samples. Jour. Ind. Hyg., vol. 21, no. 9, pp. 464-468, Nov. 1939.

Belknap, Elston L.: Clinical Studies on Lead Absorption in the Human. III. Blood Pressure Observations. Jour. Ind. Hyg., vol. 18, no. 7, pp. 380-390, Sept. 1936.

Bloomfield, J. J., and Isbell, H. S.: The Presence of Lead Dust and Fumes in the Air of Streets, Automobile Repair Shops, and Industrial Establishments of Large Cities. Jour. Ind. Hyg., vol. 15, no. 3, pp. 144-149, May 1933.

Blumgart, Herrman L., M. D.: Lead Studies: VI. Absorption of Lead by the Upper Respiratory Passages. Jour. Ind. Hyg., vol. 5, no. 5, pp. 153-158, Sept. 1923.

Brown, Lieutenant Commander, E. W., (M. C.), U. S. N.: A Study of Lead Poisoning Among Oxyacetylene Welders in the Scrapping of Naval Vessels. Jour. Ind. Hyg., vol. 8, no. 3, pp. 113-140, March 1926.

Clifford, P. A.: Report of (the Determination of) Lead (in Foods). Jour. Assoc. Agr. Chem., vol. 21, pp. 212-218, 1938.

Craig, D. Norman, and Vinal, George W.: Solubility of Lead Sulfate in Solutions of Sulfuric Acid, Determined by Dithizone with a Photronic Cell. Jour. Research Natl. Standards 22, pp. 55-70, 1939, (Research Paper, no. 1165).

Crawford, B. L., Stewart, H. L., Willoughby, C. E., and Smith, F. L.: Distribution of Lead in the Cat After Intravenous Injection of a Colloidal Lead Preparation, and the Effect of Irradiation on this Distribution. Am. Jour. Cancer, vol. 33, pp. 401-422, 1938.

Davis, C. M., M. D.: Lead Poisoning in a Golf Professional: Case Report. Jour. Ind. Hyg., vol. 5, no. 7, pp. 253-254, Nov. 1923

Dean, Archibald A., M. D., Dr. P. H.: An Epidemic of Lead Poisoning caused by the sandpapering of Automobile Bodies. Jour. Ind. Hyg., vol. 6, no. 6, pp. 245-250, Oct. 1924.

Dolowitz, D., Fazekas, J. F., and Himwich, H. D.: The Effect of Lead on Tissue Metabolism. Jour. Ind. Hyg., vol. 19, no. 2, pp. 93-94, Feb. 1937.

Fairhall, Lawrence T., A. M., Ph. D., and Shaw, Charlotte P., B. S.: The Deposition of Lead Salts, with a Note on the Solubilities of Di-Lead Phosphate in Water at 25°C. and of Di-Lead and Tri-Lead Phosphates in Lactic Acid at 25°C. Jour. Ind. Hyg., vol. 6, no. 4, pp. 159-168, Aug. 1942.

Fairhall, Lawrence T.: The Lead Content of Evaporated Milk. Jour. Ind. Hyg., vol. 19, no. 9, pp. 491-497, Nov. 1937.

Fairhall, Lawrence T.: Note on the Accuracy of Lead Analyses. Jour. Ind. Hyg., vol. 15, no. 5, p. 289, Sept. 1933.

Fairhall, Lawrence T.: The Solubility of Various Lead Compounds in Blood Serum. Jour. Biol. Chem., vol. 60, pp. 481-484, July 1924.

Fine, J.: The Phagocytosis of Lead Compounds and Their Influence on the Activity of the Leukocyte. Jour. Ind. Hyg., vol. 5, no. 5, pp. 138-144, Aug. 1923.

Flinn, Frederick B., and Smith, Adelaide Ross: The Effect of Viosterol on the Excretion of Lead. Jour. Ind. Hyg., vol. 15, no. 3, pp. 156-159, May 1933.

Grant, R. Lorimer, Calvery, Herbert O., Laug, Edwin P., and Morris, Herman J.: The Influence of Calcium and Phosphates on the Storage and Toxicity of Lead and Arsenic. Jour. Pharmacol., vol. 64, pp. 446-457, 1938.

Hamilton, Alice, M. D.: Lead Poisoning in American Industry. Jour. Ind. Hyg., vol. 1, no. 1, pp. 8-21, May 1919.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 20-63.

Hanzlik, P. J., and Presho, E.: Comparative Toxicity of Inorganic Lead Compounds and Metallic Lead for Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 123-129, March 1923.

Hanzlik, P. J., and Presho, E.: Comparative Toxicity of Metallic Lead and Other Heavy Metals for Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 145-150, March 1923.

Hanzlik, P. J., and Presho, E.: Therapeutic Efficiency of Various Agents for Chronic Poisoning by Metallic Lead in Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 131-143, March 1923.

Harrold, G. C., Meek, S. F., and Holden, F. R.: A Practical Method for the Rapid Determination of Lead When Found in the Atmosphere. Jour. Ind. Hyg., vol. 18, no. 10, pp. 724-732, Dec. 1936.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacobs: An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetraethyl Lead. Part I. Jour. Ind. Hyg., vol. 16, no. 2, pp. 100-128, March 1934.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetraethyl Lead. II. The Occupational Lead Exposure of Filling Station Attendants and Garage Mechanics. Jour. Ind. Hyg., vol. 18, no. 1, pp. 42-68, Jan. 1936.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: Lead Absorption and Excretion in Certain Lead Trades. Jour. Ind. Hyg., vol. 15, no. 5, pp. 306-319, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: Lead Absorption and Excretion in Relation to the Diagnosis of Lead Poisoning. Jour. Ind. Hyg., vol. 15, no. 5, pp. 320-340, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. I. Lead Absorption and Excretion in Primitive Life. Jour. Ind. Hyg., vol. 15, no. 5, pp. 257-272, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. II. Lead Absorption and Lead Excretion in Modern American Life. Jour. Ind. Hyg., vol. 15, no. 5, pp. 273-288, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. III. The Sources of Normal Lead Absorption. Jour. Ind. Hyg., vol. 15, no. 5, pp. 290-300, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. IV. Lead Absorption and Excretion in Infants and Children. Jour. Ind. Hyg., vol. 15, no. 5, pp. 301-305, Sept. 1933.

Key, J. A.: Lead Studies. IV. Blood Changes in Lead Poisoning in Rabbits: Stippled Cells. Am. Jour. Physiol., vol. 70, p. 86, Sept. 1942.

Klein, C. A.: Constructive Industrial Hygiene in the India Rubber Industry. Jour. Soc. Chem. Indust., vol. 41, no. 15, pp. 325R-328R, Aug. 15, 1922.

Klein, C. A.: The Prevention of Lead Poisoning in Industry. Part I. The India Rubber Industry. (Methods of Concentration of Controlled Risk). Jour. Ind. Hyg., vol. 8, no. 7, pp. 296-299, July 1926.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 412-470.

Kogan, Dr. B., and Smirnowa, Dr. L.: Changes in the Blood Picture Under the Influence of Lead, and Their Importance in the Differential Diagnosis. Jour. Ind. Hyg., vol. 9, no. 10, pp. 435-452, Oct. 1927.

Krafka, Joseph Jr., M. D.: The Effect of Repeated Leading on the Blood Picture in Guinea-Pigs. Jour. Ind. Hyg., vol. 17, no. 1, pp. 13-17, Jan. 1935.

Krans, Edward W., and Ficklen, J. B.: A Colorimetric Method for the Detection and Estimation of Small Amounts of Lead. Jour. Ind. Hyg., vol. 13, no. 4, pp. 140-143, April 1931.

Lane, Ronald E., M. B. (Lond.), M. R. C. P. (Lond.), and Lewy, F. H., M. D. (Philadelphia, Pa.): Blood and Chronaximetric Examination of Lead Workers Subjected to Different Degrees of Exposure: A Comparative Study. Jour. Ind. Hyg., vol. 17, no. 3, pp. 79-92, May 1935.

Lane, Ronald E.: Lead Burning: A Report Upon an "Exhausted Blow Pipe." Jour. Ind. Hyg., vol. 18, no. 7, pp. 391-400, Sept. 1936.

Lane, Ronald E.: The Prevention of Industrial Plumbism. Lancet, pp. 206-211, July 25, 1936.

Lane, Ronald E., M. B., B. S. (Lond.), M. R. C. P. (Lond.): The Role of Punctate Basophilia in the Control of Industrial Plumbism. Jour. Ind. Hyg., vol. 13, no. 8, pp. 276-284, Oct. 1931.

Leake, J. P.: Text of Full Report of Investigation of Health Hazards from Tetra-Ethyl Lead Gasoline. Treasury Department, U. S. Pub. Health Ser., p. 61—and 57 tables, 1926, (bound separately).

Lewy, F. H., M. D.: The Application of Chronaximetric Measurement to Industrial Hygiene Particularly to the Examination of Lead Workers. Jour. Ind. Hyg., vol. 17, no. 3, pp. 73-78, May 1935.

McCord, C. P., Minster, D. K. and Rehm, M.: The Basophilic Aggregation Test in Lead Poisoning. Jour. Am. Med. Assn., vol. 82, pp. 1759-1763, May 31, 1924.

McKail, David, M. D., D. P. H.: Prophylaxis in Industrial Lead Poisoning. Jour. Ind. Hyg., vol. 8, no. 2, pp. 74-77, Feb. 1926.

McNair, L. C., and Price, C. W.: The Effect of Rubbing Down and Scraping by Dry Process of Lead Painted Surfaces of Iron and Steel Structures. Jour. Ind. Hyg., vol. 11, no. 6, pp. 175-181, June 1929.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 147-198.

Makaritschewa, Anna, and Glagolewa, Tatiana: Ulcers of the Stomach in Lead Workers. Jour. Ind. Hyg., vol. 16, no. 4, pp. 201-202, July 1934.

Mayers, May R., M. A., M. D.: Lead Absorption and Compensation. Jour. Ind. Hyg., vol. 11, no. 4, pp. 124-138, April 1929.

Mayers, May R., M. A., M. D.: Lead Anemia. Jour. Ind. Hyg., vol. 8, no. 5, pp. 222-231, May 1926.

Mayers, May R., M. A., M. D.: A Study of the Lead Line, Arteriosclerosis, and Hypertension in 381 Lead Workers. Jour. Ind. Hyg., vol. 9, no. 6, pp. 239-250, June 1927.

Minot, A. A., Ph. D.: Lead Studies 5. A. The Distribution of Lead in the Organism After Absorption by the Gastro-Intestinal Tract. Jour. Ind. Hyg., vol. 6, no. 4, pp. 125-136, Aug. 1924.

Minot, A. S., Ph. D.: Lead Studies 5. B. The Distribution of Lead in the Organism After Absorption by the Lungs and Subcutaneous Tissue. Jour. Ind. Hyg., vol. 6, no. 4, pp. 137-148, Aug. 1924.

Minot, A. S., Ph. D., and Aub, J. C., M. D.: Lead Studies 5. C. The Distribution of Lead in the Human Organism. Jour. Ind. Hyg., vol. 6, no. 4, pp. 149-158, Aug. 1924.

Moliter, P., Arnoldson, M., and Hausser, G.: Detection of Lead Poisoning in a Motor Car Factory. Arch. Mal. Profes., vol. 1, pp. 124-128, 1938.

Newman, Bernard J., McConnell, William J., Spencer, Octavius M., and Phillips, Frank M.: Lead Poisoning in the Pottery Trades. U. S. Public Health Service, Public Health Service, Public Health Bulletin No. 116, May 1921, pp. 220 with Index. Washington: Government Printing Office, 1921.

Norris, C., and Gettler, A. O.: Poisoning by Tetra-Ethyl Lead: Postmortem and Chemical Findings. Jour. Am. Med. Assn., vol. 85, pp. 818-820, Sept. 12, 1925.

Nye, L. J. Jarvis, M. B., Ch. M.: Chronic Nephritis and Lead Poisoning. pp. 138 and Bibliography. Sydney: Angus & Robertson, Ltd., 1933.

Occupation and Health. International Labour Office, Geneva, vol. 2, pp. 99-141.

Oliver, Thomas: Saturnine Asthma: Is There Such A Malady? Lancet, vol. 203, no. 5147, pp. 907-909, Oct. 28, 1922.

Ohio Department of Health: Lead and Its Compounds, 1940.

Otto, H., and Kuhlman, F.: Gastro-Intestinal Tract, Especially Disturbance of the Intestines in Lead Poisoning. Klin. Wchnschr., vol. 18, pp. 1081-1085, 1939.

Painters' Colic: How Caused and How Best Prevented. Reprinted from Home Office Form 394. H. M. Stationery Office, June 1923, p. 2.

Pedley, Frank G., M. D., Dr. P. H.: The Effects of Lead on the Vision: A Case of Subhyaloid Hemorrhage. Jour. Ind. Hyg., vol. 12, no. 10, pp. 359-363, Dec. 1930.

Perlman, J. L.: Rapid Colorimetric Determination of Lead in Maple Sirup. Ind. Eng. Chem. Anal. Ed. 10, pp. 134-135, 1938.

Resin, Paul F., and Drinker, Philip: Volatilization of Lead Below 800°C. Jour. Ind. Hyg., vol. 21, no. 9, pp. 461-463, Nov. 1939.

Sautter, Albert C.: Encephalopathy with Ocular Complications Probably Due to Lead Poisoning. Am. Jour. Ophth., vol. 5, no. 6, pp. 468-470, June 1922.

Sellers, Arthur, M. D., D. P. H.: Blood Changes in Lead Workers. Jour. Ind. Hyg., vol. 2, no. 10, pp. 361-367, Feb. 1921.

Sellers, Arthur, M. D., D. P. H.: A Contribution to the Study of Punctate Basophilia in Lead Workers. Jour. Ind. Hyg., vol. 7, no. 4, pp. 145-154, April 1925.

Sharpe, N. C., A. B., M. B.: Report on an Investigation to Determine the Hazard to the Health of Operators Using the Spraying Machine for Painting: The Risk of Lead Poisoning. Jour. Ind. Hyg., vol. 3, no. 12, pp. 378-386, April 1922.

Shie, M. D.: Lead Poisoning: Its Symptomatology and Diagnosis. Jour. Am. Med. Assn., vol. 83, pp. 580-583, Aug. 23, 1924.

Shields, J. B., Mitchell, H. H., and Ruth, W. A.: The Metabolism and Retention of Lead in Growing and Adult Rats. Jour. Ind. Hyg., vol. 21, no. 1, pp. 7-23, Jan. 1939.

Shiels, D. O.: The Ratio of Large to Small Lymphocytes in Persons Exposed to a Lead Hazard. Med. Jour. Australia, no. 25, pp. 847-849, June 20, 1936.

Straube, G., and Beck, H.: Micro-analytic Detection of Lead in Body Liquids. II. Klin. Wchnschr., pp. 356-360, 1939.

Surgeon Gen. Committee, U. S. Pub. Health Service, Washington, D. C.: Report on Tetraethyl Lead. Jour. Ind. Hyg., vol. 8, no. 5, pp. 248-256, May 1926.

Tebbens, B. D.: Atmosphere Lead Contamination from High Temperature Lead Baths. Jour. Ind. Hyg., vol. 19, no. 1, pp. 6-11, Jan. 1937.

Teleky, Ludwig, M. D.: German Literature On the White Lead Question, 1921. Jour. Ind. Hyg., vol. 4, no. 3, pp. 100-105, July 1922.

Teleky, L.: A Note on Blood Pressure in Lead Poisoning. Jour. Ind. Hyg., vol. 19, no. 1, pp. 1-5, Jan. 1937.

Vigdortchik, N. A.: Lead Intoxication in the Etiology of Hypertonia. Jour. Ind. Hyg., vol. 17, no. 1, pp. 1-6, Jan. 1935.

Vogt, E. C.: Roentgen Sign of Plumbism: Lead Line in Growing Bone. Am. Jour. Roentgenol., vol. 24, p. 550, Nov. 1930.

Weller, C. V.: Lead Meningo—Encephalopathy. Ann. Clin. Med., vol. 3, p. 604, March 1925.

Wright, Wade, M. D., Sappington, Clarence O., M. D., and Rantoul, Eleanor: Lead Poisoning from Lead Piped Water Supplies. Jour. Ind. Hyg., vol. 10, no. 7, pp. 234-252, Sept. 1928.

MANGANESE

Chemical Formula and Synonyms:

(Manganese) Mn.
(Pyrolusite) MnO_2, manganese dioxide, black.

A. Principal Properties:

1. Manganese
   a. A reddish-gray or silvery, brittle metalic element. Sp. gr. 7.2 at 20°C.; m.p. 1,260°C. Decomposes in water.
2. Pyrolusite
   a. Iron black to dark steel-gray or bluish mineral; streak, black or bluish-black; luster, metallic or dull. Sufficiently soft to soil the fingers. Contains 63.2% manganese. Sp. gr. 4.73 to 4.86; hardness 2 to 2.5. Soluble in hydrochloride acid.

B. Principal Industrial Uses:

1. Manganese
   a. Manufacturer of steel (deoxidizer); iron, copper and aluminum alloys; chemicals.
2. Pyrolusite
   a. Preparation of manganese.

C. Poisoning—Systemic

1. Modes of Entry
   a. Inhalation
2. Symptoms
   a. The symptoms of manganese poisoning are generally associated with inhalation of its oxides and primarily concerned with affections of the central nervous system. The effect of manganese is said to be cumulative and the symptoms usually appear only after several months of exposure. They are evidenced by a peculiar slapping gait, weakness in legs and tremors of the whole body or extremities. Other symptoms frequently observed are mask-like face, impulsive and uncontrollable laughter, disturbances of speech, languor and sleepiness, cramps and stiffness of the muscles, propulsion and retropulsion, and exaggeration of the reflexes.

   It is said that manganese, unlike lead, produces no life shortening degenerations. Seriously poisoned victims are life long cripples and often unfit for any gainful employment. The metal apparently makes a very definite attack upon some non-vital portion of the neuro-muscular system, destroys it thoroughly if there has been sufficient exposure, and leaves the victims relatively well in every other respect.

D. Sanitary Corps Officer:

1. Control measures.

SELECTED REFERENCES

Bickert, F. W.: The Health of Workers in Manganese Ore Mills. Arch. f. Gewerbépathol. u. Gewerbehyg., vol. 4, pp. 647-688, 1933.

Bryan, A. W.: Chronic Manganese Poisoning: Report of a Case. (Society Transactions). Arch. Neurol. and Psychiat., vol. 37, pp. 1448-1449, June 1937.

Buttner, H. E., and Lentz, E.: Possibility of Manganese Poisoning in Pyrolusite Mines. Arch. Gewerbepath. Gewerbehyg., vol. 7, pp. 672-684, 1937.

Charles, J. R.: Three Cases of Manganese Poisoning. Jour. Neurol. and Psychopath., vol. 3, pp. 262-268, Nov. 1922.

Crouzon, O., and Desoille, H.: Nerve Damage in Industrial Manganese Poisoning. Paris Med., vol. 26, pp. 361-365, 1936.

Dairs, George G., M. D., and Huey, Walter B., M. D.: Chronic Manganese Poisoning: Two Cases. Jour. Ind. Hyg., vol. 3, no. 8, pp. 231-238, 1921. Dragonetti, Michele: Chronic Intoxication with Manganese. Rass. Med. Applicata Lavoro Indust., vol. 9, pp. 94-106, 1938.

Dragonetti, M.: Chronic Manganese Poisoning, a Study of One Case. Rass. di Med. Appl. Lavoro Indust., vol. 9, pp. 94-106, April 1938.

Edsall, Daird L., M. D., Wilbur, F. P., M. D., and Drinker, Cecil L., M. D.: The Occurrences, Course and Prevention of Chronic Manganese Poisoning. Jour. Ind. Hyg., vol. 1, no. 4, pp. 183-193, Aug. 1919.

Ehrismann, O.: Studies on the Absorption of Dust in the Respiratory Passages. II. Absorption of Manganese Dioxide Dust. Ztschr. f. Hyg. u. Infection-skrank., vol. 117, p. 662, 1935.

Factory and Workshop Order: Notification of Manganese Poisoning. (Great Britain) July 1936.

Feil, Andre: Manganese Poisoning in Industry. Presse Med., vol. 45, pp. 1593-1594, 1937.

Gaylo, R. F., Jr.: Manganese Poisoning and Its Effect on the Central Nervous System. Report of Six Cases. Jour. Am. Med. Assn., vol. 85, pp. 2008-2011, Dec. 26, 1925.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 92-96.

Jotten, K. W.: Pulmonary Injury from Manganese and Thomas Slag Dust. Deutsch. Med. Wchnschr., vol. 65, pp. 409-410, 1939.

Jotten, K. W., and Reploh, H.: Experimental Study of Pulmonitis Produced by the Inhalation of Dust Containing Manganese. Rass. med. applicata lavoro indust. vol. 9, pp. 395-398, 1938.

Jotten, K. W., Reploh, H., and Hagemann, G.: Experimental Investigation of Pneumonia Caused by Manganese and Its Relation to Pneumonia from Thomas Slag. Arch. f. Gewerbepath., vol. 9, pp. 314-336, 1939.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 589-592.

Loebe, C. V.: Manganese Poisoning. Jour. f. Psychol. u. Neurol., vol. 47, pp. 220-221, 1936.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 134-139.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 158-161.

Ohio Department of Health, Manganese, 1940.

Polonovski, M., and Briskas, S. B.: Hemopoieric Action of Zinc, Manganese and Nickel. Compt. rend. Soc. Biol., vol. 129, pp. 493-496, 1938.

Rastelli, G.: Complement Formation Ability in Manganese Poisoning. Med. del lavero, vol. 29, pp. 353-363, 1939.

Scander, A., and Sallam, H. A.: A Report on Eleven Cases of Chronic Manganese Poisoning. Jour. Egyptian Med. Assoc., vol. 19, pp. 57-62, 1936.

Schwartz, L., and Pagols, J.: Experiments on the Early Diagnosis of Industrial Manganese Poisoning. Arch. f. Hyg., vol. 92, pp. 77-84, June, 1923.

Schwarz, L.: Industrial Manganese Poisoning. Samm. v. Vergiftungsf., vol. 7, p. 5, 1936.

Sharpe, N. C.: Some Clinical Aspects of Industrial Poisoning. Manganese Poisoning in Storage Battery Workers. Pub. Health Jour., vol. 14, pp. 110-118, March 1923; pp. 172-174, April 1923.

Trendtel, F.: Manganese Poisoning. Monatsschr. f. Unfallheil., vol. 42, pp. 69-84, 1936.

Voss, H.: The Occurrence of Occupational Manganese Poisoning in the Steel Industry (Apropos of Manganese Poisoning in a Grinder of Ferro-Manganese). Arch. f. Gewerbepath., vol. 9, pp. 453-463, 1939.

Factory and Workshop Order: Notification of Manganese Poisoning. (Great Britain) July 1936.

MERCURY: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Mercury) Hg, hydrargyrum, quicksilver.
(Mercury fulminate) $Hg(CNO)_2$, fulminate of mercury.
(Copper Amalgam)
(Mercury Bichloride) Mercuric chloride, corrosive sublimate.

A. Principal Properties:

1. Mercury
   a. A silvery, liquid, metallic element, sometimes found native; poisonous. Sp. gr. 13.5953; m.p. —38.85°C., b.p. 357.33°C. Soluble in nitric acid, insoluble in water, alcohol and ether; wt. per liter of vapor, 8.34 gr.
2. Mercury fulminate
   a. Dark brown, crystalline powder; explodes when dry under the slightest friction or shock; must be kept moist until used. Sp. gr. 4.42; m.p.: explodes. Soluble in alcohol, ammonium hydroxide and hot water; slightly soluble in cold water.
3. Copper Amalgam
   a. A solution of copper in mercury of variable composition.
4. Mercury Bichloride
   a. White rhombic crystals; sp. gr. 5.44; m.p. 277°C.; b.p. 304°C. Soluble in water, alcohol and ether.
5. Important Compounds
   a. Mercuric acetate; mercuric-ammonium chloride, mercuric benzoate, mercuric bromide, mercuric chloride, mercuric cyanide, mercuric iodide, mercuric nitrate, mercuric oleate, mercuric oxide, red; mecuric oxide, yellow; mercuric-potassium cyanide, mercuric-potassium iodide, mercuric salicylate, mercuric sulfate, mercuric sulfide, black; mercuric sulfide, red; mercuric sulfocyantae, mercurous sulfate, mercury fulminate.

B. Principal Industrial Uses:

1. Mercury
   a. Mercury salts; thermometers; medicine; mirror manufacture; mercury vapor lamps; amalgams; extraction of gold and silver from their ores; physical and chemical apparatus; catalyst; production of bulminate and vermillion; electric rectifiers; pharmacy; cathode in electrolytic chemical processes; felt manufacture; boiler compounds; cosmetics.
2. Merury Fulminate
   a. Manufacture of caps and detonators for producing explosions for military, industrial and sporting purposes.
3. Copper Amalgam
   a. Formerly used as dental fillings; used in manufacture of brushes for electric generators and motors.
4. Mercury Bichloride
   a. Medicine; antiseptic; preservative for skins, furs and wood.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation, ingestion and absorption through the skin and subcutaneous tissues. The chief mode of entrance of mercury vapor and dust is by inhalation, of the liquid compounds and solutions of mercury by ingestion, and absorption through the skin or possible inhalation of vapors.
2. Symptoms
   a. (Mercury) Industrial mercury poisoning is characterized by three cardinal features; one or more of which may be present simultaneously; inflammation of the mouth (stomatitis), psychic irritability (erethism), and muscular tremors ("Hatters shakes"). Inflammation of the mouth and psychic irritability are stated to be more evident in the acute cases in which mercury is absorbed over a short period; tremor may occur more frequently in chronic cases in which absorption has occurred slowly over a long period of time. Symptoms may include metallic taste, sensation of abnormal dryness of mouth and throat, stomatitis, inflammation and softness of the gums, loosening of the teeth; salivation, pain on chewing, blue line on gums, and foetid breath. Psychic changes are usually present such as nervous timidity, irritability, discouragement, depression, ease of embarrassment, blushing, desire for solitude, vague fears, as of ridicule or criticism, fits of unreasonableness, impatience, inability to take orders; apathy, lack of interest, loss of memory and self-confidence, despondency, and even suicidal tendencies. There is an intentional tremor which is vibratory, intermittent, in small equal strokes and rhythmical, worse with efforts to control it, on unusual movements or when movements are observed; it disappears in sleep. The tremor attacks first the eyelids, tongue, fingers, then the voluntary muscles and is usually symmetrical. The central nervous system manifestations of mercurialism are due, in the opinion of some authorities, to a diffuse encephalopathy with predominance of symptoms referable to the cerebral centers most affected.

   In the more severe cases there may be found headache, vertigo, and constipation, abdominal cramps or distension, weakness and exhaustion, and pain in the muscles, bones and joints. Usually in industrial mercurialism gastro-intestinal symptoms are not prominent, and there is not much if any involvement of the kidneys, apparently they can eliminate mercury for a long period without damage, and urinary changes may be slight or absent in the slow chronic form of poisoning. An increased incidence of albumin in the urine has been reported; lymphocytosis of slight degree is also stated to occur.

   Inflammation may occur at the points of excretion of mercury, thus nephritis, colitis, gastric and duodenal ulcer have been reported as a sequelae of severe poisoning. Also inflammation may occur at the openings of salivary ducts or other points of excretion. It is believed that mercury ingested or absorbed as dust would form in the blood chloro-albuminate or oxychloro-albuminate while mercury inhaled as vapor would circulate as such in the blood for some time.

   b. (Mercury Fulminate) Controversy exists whether symptoms of exposed workers are due to this substance or another. Fulminate workers may show various forms of eczema and dermatoses which are particularly important. Also there may be present stomatitis, salivation, blackened, brittle or decayed teeth, inflammation of the gums, conjunctivitis, blepharitis, digestive disturbances, diarrhea, menstrual disturbances, headache, insomnia, tremors, cachexia, and depression. Poisoning may result from mercury fumes when the mercury detonates as has occurred in shooting galleries, etc. The symptoms observed are stomatitis, nausea, vomiting and colic.

c. (Copper Amalgam) Symptoms produced by this substance are due to the dust or vapor arising in its use. The symptoms are similar to those produced by mercury, the severity of them depending on the amount of dust or vapor present.

d. (Mercury Bichloride) The increased solubility of mercury bichloride makes it more toxic than free mercury. Poisoning may occur accidentally or with suicidal intention. Symptoms of acute poisoning due to the coagulation, irritation and superficial corrosive action may occur in a few minutes. There may be a burning sensation in the throat, astringent metallic taste, salivation, great thirst, abdominal distress, pain and vomiting in about five minutes. There is a gray ashy discoloration with a white coating forming in the mouth and pharynx, with edema of glottis. Temperature may be febrile or subnormal, cold sweat with weak irregular pulse. Diarrhea may occur within two hours with a liquid and blood stained stools. The urine is scanty or depressed, cells and albumin being present; and anuria may occur with a rise in the non-protein nitrogen of the blood. The salivation, stomatitis, glossitis and gingivitis with severe abdominal pains may persist several days.

3. Laboratory Procedures.
   a. A simplified procedure for the determination of mercury in urine: A 50 cc. portion of the urine is ashed in a 500 cc. Kjeldahl by means of potassium permanganate and sulfuric acid. The mercury is separated by means of diphenylthiocarbazone and the intensity of color compared with that of known standards. The ordinary chemically pure reagents were found to be satisfactory and the blanks were negligible. The time required for a series of five determinations is about three hours. The simplified procedure is more rapid than the Winkler method and equally accurate.
   b. Increase of lymphocytes in the differential blood count.
   c. Urinary changes may be present in the mercurial poisoning.
4. Toxic Concentrations.
   a. Toxic dose of mercury vapor cannot be stated with certainty but Gothlin concludes that 0.4 to 1 mg. of mercury absorbed daily would set up a gradual chronic poisoning after some months.

D. Sanitary Corps Officers:

1. Rigid control measures where Mercury vapors are being used.
2. The proper use and care of masks.

E. Medical Control:

1. Pre-employment placement.
   a. Youth, female sex, alcoholism and infection appear to increase the susceptibility to mercury poisoning, and therefore should not be exposed to this hazard.

SELECTED REFERENCES

Busch, E.: Action of Occupational Mercury Poisoning. Arch. f. Gewerbepathol. u. Gewerbehyg., vol. 5, pp. 199-200, 1934.

Duschak, L. H., and Schuette, C. N.: The Metallurgy of Quicksilver. U.S. Bur. Mines, Bull. 222, pp. 173, 1925.

Eisenbarth, H.: Mask Protection against Mercury Vapor. Gasmaske, vol. 6, pp. 156-157, 1934.

Fellinger, K., and Schweitzer, F.: Vascular Diseases after Mercury Poisoning. Arch. f. Gewerbepath., vol. 9, pp. 269-275, 1938.

Francioni, G.: Histological Lesions of the Central Nervous System in Experimental Poisoning by Mercury Vapor. Rass. di. Med. Appl. al Lav. Indust., vol. 6, pp. 110-116, April 1935.

Fraser, A. M.: The Determination of Mercury in Air and Urine. Jour. Ind. Hyg., vol. 16, pp. 67-76, 1934.

Fraser, A. M., Menville, K. I., and Stehle, R. R.: Mercury Laden Air: Toxic Concentration, Proportion Absorbed, and Urinary Excretion. Jour. Ind. Hyg. vol. 16, pp. 77-91, 1934.

Friederich and Buhr: Mercury Poisoning and its Chemical Detection. Suddeutsch. Apoth. -Ztg., vol. 68, pp. 702-703, 1928; Chem. Zentrlbl., part 2, p. 2739, 1928.

Gelman, I., and Derirz, G.: The Problem of the Fate of Mercury Fumes and Mercurial Compounds in the Organism. Jour. Ind. Hyg., vol. 19, no. 6, pp. 215-224, June 1937.

Grosskopf, K.: Detection of Small Quantities of Mercury in Air. Dragerheft, no. 191, pp. 3589-3591, 1937.

Guiljarowsky, W., and Winokuroff, A.: The Clinical Picture and Pathology of Mercury Poisoning. Ztschr. f. d. ges. Neurol. u. Psychiat., vol. 121, p. 1, 1929.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 72-81.

Hamilton, Alice, M. D.: Industrial Diseases of Fur Cutters and Hatters. Jour. Ind. Hyg., vol. 4, no. 5, pp. 219-234, Sept. 1922.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 177-182.

Industrial Mercurialism. Report of an Investigation. Ohio Department of Health, Columbus, Ohio, 1940, 16 pp.

Jacobsohn, William, M. D.: Mercurial Poisoning in the Manufacture of Clinical Thermometers. Jour. Ind. Hygiene, vol. 2, no. 5, pp. 193-196, Sept. 1920.

Johnstone, B. I.: Acute Mercury Poisoning Report of Twenty-One Gases with Suggestions for Treatment. Canadian Med. Assn. Jour., vol. 24, pp. 500-507, April 1931.

Kam, A. J. H.: The Determination of Small Amounts of Mercury. Versl. Volksgzndh., pp. 585-601, 1931.

Killian, J. A.: The Blood Chlorides in Mercuric Chloride Nephritis. Jour. Lab. and Clin. Med., vol. 7, pp. 129-133, 1921.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son And Co., 1924, pp. 471-494.

Koch, W.: Nephrotic Contracted Kidney after Acute Mercury Poisoning. Med. Klin., vol. 33, pp. 1559-1563, 1937.

Koiransky, B. B., and Benediktova, E. J.: Extensor Weakness in Workers with Mercury. Zentralbl. f. Gewerbehyg., N. S., vol. 8, pp. 169-173, July 1931.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 199-219.

Majer, V.: Poisoning Effect of Mercury Vapors. Chem. Abzor., vol. 10, pp. 51-53 (in English, p. 53), 1935.

Mallison, H.: Kraemer-Sarnow Method (For Determining the Softening Point of Pitch) and Mercury Poisoning. Ztschr. f. angew. Chem., vol. 41, pp. 839-840, 1928.

Mercury. Analysis of Factory Inspection Reports, 1920-1922, III. Internat. Labour Rev., vol. 13, pp. 541-546, April 1926.

Minot, Annie Stone: Estimations of Mercury in Hatters' Fur and in Felt. Jour. Ind. Hyg., vol. 14, no. 6, pp. 253-255, Oct. 1922.

Moller, L.: Mercurial Stomatitis as an Occupational Disease. Klin. Wchnschr., no: e, 1923.

Monakow, P. v.: Kidney Function after Damage by Heavy Metals (Mercury, Kollargol) with a Note on Clinical Aspects of Bang's Disease. Deutsch. Arch. f. klin. med., vol. 183, pp. 155-170, 1938.

Muller, R., and Scheiner, K.: Comparative Histological and Quantitative Spectrographic Studies in Experimental Mercury Poisoning. Arch. expt. Patch. Pharmakol., vol. 180, pp. 718-730, 1936.

Neal, P. A., and Jones, R. R.: Chronic Mercurialism in the Hatters' Fur Cutting Industry. Jour. Am. Med. Assn., vol. 110, pp. 337-342, Jan. 29, 1938.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 211-227.

Ohio Department of Health, Mercury and its Compounds, 1940.

Pinkus, G.: The Dangerous Nature of Mercury Vapor. Ztschr. f. angew. Chem., vol. 39, pp. 787-788, 1926.

Podrouzek, R.N.V.: Mercury Poisoning. Chem. Listy., vol. 22, pp. 529-Wchnschr., vol. 70, p. 1409, Nov. 23, 1923.

Rakusin, M. A., and Nesmejanow, A. N.: Antidote to Mercuric Chlorid. Wchnschr., vol. 70, p. 1409, Nov. 23, 1923.

Rakusin and Nesmejanow: Charcoal and Metal Salts. Munchen. Med. Munchen. med. Wchnschr., vol. 71, p. 1168, Aug. 22, 1924.

Ruska, J.: Observations on Mercury Poisoning by Arabic Alchemists and Physicians. Ztschr. f. angew. Chem., vol. 39, p. 790, 1926.

Sayers, R. R.: Mercury Poisoning. U. S. Bur. Mines. Rep. Investigations No. 2354, 1922.

Schrieber, N. E., Sollmann, T., and Booth, H. S.: The Determination of Traces of Mercury. III. The Quantitive Determination of Mercury in Urine and Feces and the Influence of Medication. Jour. Am. Chem. Soc., vol. 50, pp. 1620-1625, June 1928.

Shapiro, P. F.: Mercury Poisoning: Its Clinical Difficulties and its Pathogenesis. Report of Two Cases of Brown Sublimate Kidney. Jour. Lab. and Clin. Med., vol. 15, pp. 961-972, July 1930.

Stock, A.: Carbon-Iodine as Protection for Mercury Vapor Poisoning. Angew. Chem., vol. 47, p. 64, Jan. 1934.

Teleky, L.: Does Lead Poisoning or Mercury Poisoning Persist, Relapse, or Recur? Arch. f. Gewerbepath., vol. 5, pp. 132-157, 1933.

Wolf, L.: The Dangerous Nature of Mercury Vapor. Ztschr. f. Angew. Chem., vol. 39, pp. 789-790, 1926.

METAL FUME FEVER

A. Principal Properties:

1. Fumes signify liquid or solid particles from about 0.2 micron to 1 micron in diameter, formed by physiochemical reaction such as distillation, condensation and combustion.
2. Fumes have also been defined as solid particles generated by condensation from the gaseous state, generally after volatilization from molten metals, etc., and often accompanied by a chemical reaction such as oxidation.
3. One of the characteristics of fumes is flocculation.

B. Principal Uses:

1. In industries where metal, such as zinc, is heated in an oxidizing atmosphere to a temperature near its boiling point (zinc 930° C), for example, in the founding of brass and oxyacetylene welding of galvanized iron.
2. In the melting, stirring and casting of brass, or in any operation where zinc is heated to temperatures causing volatilization of the metal and consequent liberation of fumes, which oxidize on contact with air.
3. In steel manufacture resulting from the fusing of manganese; from cadmium in spelter works, or during the melting of cadmium ingots, as well as from smelting and electrolytic recovery of zinc; from antimony, in the printing trade, and from the vulcanization of rubber.
4. The metallic oxides commonly given off in the form of fumes or dust in welding processes may originate from several sources—from the materials welded, from the coatings of the surfaces being welded or cut, from the coatings of the electrodes used in arc welding, or from the flux rods. Some of these metallic and mineral substances are as follows: Iron, Zinc, Lead, Brass, Bronze, Copper, Nickel, Arsenic, Cadmium, Aluminum, Manganese, Phosphorus, Selenium and Silicon.
5. In the process of metallization. This operation is where molten metal is projected by compressed air against the surface to be coated and the metallic vapors may condense and oxidize.

C. Poisoning—Systemic:

1. Mode of entry:
   a. Inspiration.
   b. Questionable through the skin.
2. Symptoms:
   a. Acute: Usually come on some hours after exposure; they are aggravated by chilling of the body and are more marked in the winter. After the individual leaves work, a dryness of the throat is first noticed, followed by a dry cough and a feeling of weight in the chest. The temperature then rises and is accompanied by sweating. At this stage a leucocytosis will be found; increase of pulse rate and often an elevation of the blood pressure. The symptoms usually subside in about twenty-four (24) hours. Metal fume fever is often diagnosed as influenza or pneumonia.
   b. One of the characteristic features of metal fume fever is that "immunity" to those attacks is acquired by the workers. This immunity is rapidly lost. Hence, cases are commonest on Mondays, after holidays, and amongst new workers.
3. Pathology:
   a. No authentic cases of fatal metal fume fever have been reported in the literature.
   b. Irritative process may cause broncho-pneumonia or edema of the lungs depending on type of metal fumes.
   c. Toxic concentrations—None established.

D. Sanitary Corps Officers:

1. Proper ventilation suitable for the type of metals being used.
2. Personal protective devices. Masks and respirators should be specially adapted to the material used.

E. Medical Control:

1. Preemployment placement examination.
   a. Workers who are subject to chronic bronchitis, bronchiectasis, asthma, arrested pulmonary tuberculosis or chronic heart disease, should not be exposed to fumes that may occasionally cause new workers to develop metal fume fever.
2. Treatment.
   a. Because of the temporary nature of the disease, and since the patient recovers within a relatively short time, only symptomatic treatment is necessary, viz.: rest in bed, with adequate warmth and protection from drafts. No specific treatment has been shown to be universally effective.
3. Periodic Rechecks.
   a. Investigations as to the metals causing metal fume fever show they may also, if continued, permit absorption and accumulation of toxic metals in sufficient amounts later to produce systemic metal poisoning.

SELECTED REFERENCES

Hamilton, Alice: Industrial Toxicology. Harper and Brothers, New York, 1934.

Drinker, Philip, and Hatch, Theodore: Industrial Dust. McGraw-Hill Book Co. Inc., New York, 1936.

Safir, Horia, Our Present knowledge of metal fume fever. Verof. a. d. Geb. d. Medizinalverwalt, 38: 599-627 (1932). Abst. in J. Ind. Hyg. 15: 70 (1933).

Koelsch, F.: Metal-fume fever. J. Ind. Hyg. 5: 87 (July 1923).

Drinker, Philip, Thomson, Robert M., and Finn, Jane L.: Metal fume fever. III. The effects of inhaling magnesium oxide fume. J. Ind. Hyg. 9: 187 (May 1927).

Collier, H. E.: Poisoning by metals. Birmingham Med. Rev. 11: 238-243 (September 1936).

Doig, A. T., and McLaughlin, A. I.: X-ray appearances of the lungs of electric arc welders. Lancet, 1: 771 (April 4, 1936).

New Standard Dictionary of the English Language. Funk and Wagnalls, New York, 1937.

Barreto, J. B., Drinker, Philip, Finn, Jane L. and Thomson, R. M.: Masks and respirators for protection against dusts and fumes. J. Ind. Hyg. 9: 26 (January 1927).

Drinker, Philip, Thomson, Robert M., and Finn, Jane L.: Metal fume fever. IV. Threshold doses of zinc oxide preventive measures, and the chronic effects of repeated exposurs. J. Ind. Hyg. 9: 331 (August 1927).

Copper, Brass, Zinc.: Occupation and Health Series, International Labour Office, Geneva, 1930.

Drinker, Cecil K., and Fairhall, Lawrence T.: Zinc in relation to general and industrial hygiene. Pub. Health Rep. 48: 955 (August 11, 1933).

Batchelor, Roger P. Fehnel, J. William Thomson, Robert M., and Drinker, Katherine R.: A clinical and laboratory investigation of the effect of metallic zinc, of zinc oxide, and of zinc sulphide upon the health of workmen, J. Ind. Hyg. 18: 322 (August 1926).

Health Protection of Welders. Industrial Health Section, Metropolitan Life Insurance Co., New York, p. 20.

Drinker, Katherine R., and Drinker, Philip: Metal fume fever. V. Results of the Inhalation by anímals of zinc and magnesium oxide fumes. J. Ind. Hyg. 10: 56-70 (February 1928).

Prodan, Leon: Cadmium Poisoning: II. Experimental cadmium poisoning. J. Ind. Hyg. 5: 174-195 (May 1932).

Drinker, Philip: Certain aspects of the problem of zinc toxicity. J. Ind. Hyg. 4: 177 (August 1922).

Gases and Fumes. Occupation and Health Series, International Labour Office, Geneva, 1930.

Industrial exhaust systems. Guide 1935. Chapter 21, p. 346, American Society of Heating and Ventilating Engineers, New York.

List of devices for respiratory protection approved by the U. S. Bureau of Mines, I. C. 6918, October 1936.

Sayers, R. R.: Metal Fume fever and its Preventions, Public Health Reports, vol. 53, No. 26, July 1, 1938, Pages 1080-1086.

NITROUS FUMES

Nitrous Fumes, produced in the pickling of metals, especially brass, in the combustion of explosives, and whenever nitric acid comes in contact with organic materials, such as sawdust and excelsior, represent a mixture of different oxygen derivatives of nitrogen. They generally consist of Nitrogen Oxide, Nitrogen Dioxide, Nitrogen Tetroxide, Nitrous and Nitric Acids.

1. *Nitrous Oxide*
   a. Although Nitrous Oxide may be present in Nitrous fumes when these are formed in the absence of larger quantities of oxygen or air, it is of no importance from the industrial toxicologic point of view, because at most the quantities are too small to cause the effects characteristic of this gas. It is used commercially as an anesthesia agent (Nitrous Oxide).

2. *Nitrogen Oxide*
   a. The effect of nitrogen oxide in the animal organism resembles in many respects that produced by nitrous oxide, but whereas the latter has a direct, although moderate depressant effect on the central nervous system, this has not been proven for the action of nitrogen oxide and it appears not unlikely that the phenomena observed in nitrogen oxide poisoning are solely due to anoxia caused by methemoglobinemia.
   b. In view of the comparatively rapid oxidation of nitrogen oxide to nitrogen dioxide no cases of human nitrogen oxide poisoning are reported in the literature. Hamilton (1925) stated that there is a less familiar form of nitrous fume poisoning which is rapidly fatal, causing only slight anatomical changes, so that it appears in such cases death is caused by direct action on the respiratory center. She mentions five instances of such sudden death in a nitration plant and it appears not impossible that in these nitrogen oxide was the cause of death. Similarly, a case reported by Miller (1925) may have had exposure to nitrogen oxide in addition to nitrogen dioxide because in this instance severe methemoglobinemia was also observed, leading to bilirubinemia and icterus.

3. *Nitrogen Dioxide*
   a. Nitrogen Dioxide is the most important oxide of nitrogen from the toxicologic point of view.
      (A) *Principal Properties:*
         (1) Reddish brown gas of the molecular weight 46.01 and specific gravity 1.448 at 20° C., which is distinctly soluble in water.
         (2) The polymer, nitrogen tetroxide is a colorless fluid with a peculiar sweetish, acrid odor and the molecular weight 92.02. It has been pointed out that whatever molecular form nitrogen dioxide is inhaled, it is at once altered to that prevalent at body temperature, which is a mixture of approximately 30% nitrogen dioxide and 70% nitrogen tetroxide.. Nitrogen tetroxide reacts with water with the formation of nitric and nitrous acid. Nitrogen dioxide reacts with water to produce nitric acid and nitric oxide. The nitrogen oxide found in this reaction is oxidized and converted into nitric acid. For this reason nitrous fumes represent a mixture of nitrogen oxide, nitrogen dioxide, nitrogen tetroxide and in addition, if moist, a mist of nitric and nitrous acid.
      (B) *Principal Industrial Uses:*
         (1) The fumes may be encountered in the manufacture of nitric acid and in the nitration of cellulose and other organic materials, as in the manufacture of explosives, dyes, lacquers and certain types of film and celluloid. Such fumes may also be encountered in the bleaching

of cotton and of raw silk with nitric acid; in the metal industry during pickling, during etching, and with the use of acqua regia in soldering; and also under certain conditions, in electric arc welding and with the use of an acetylene torch in small enclosures.

(2) Incidence of Nitrous Fume Poisoning in Various Operations.

Cause of Accident	*Total Number of Fatalities Reported*
Breaking of container	28
Nitration	12
Cleaning of Tank, Chamber or Tower	12
Spilling of Acid	11
Decomposition of artificial fertilizer	4
Pickling or cleaning metal	16
Manufacture of Sulfuric Acid	4
Welding in Tank or Boiler	7
Unstated or other causes	13
	107

(3) Poisonings from exposure to nitrous fumes resulting from explosions on battleships have been reported, the danger being of nitrous fumes of smokeless powder, cordite, or other high explosives, which are burned or exploded in the absence of sufficient air.

(C) *Poisoning—Systemic:*

(1) Modes of entry

a. Inhalation.

(2) Symptoms

Concerning the clinical picture of nitrous fumes poisoning, four types are distinguished.

(a) Irritant gas type.
(b) The reversible type.
(c) The shock type.
(d) The combined type.

a. The irritant gas type is characterized initially by more or less severe irritation resulting in sensation of pain, burning, choking in throat and chest, violent cough and the expectoration of yellow-tinged sputum. Immediately or later the sputum may also contain blood. These symptoms, which may be of short duration, are followed by a latent period during which the patient may feel quite comfortable and be able to pursue his work or walk home, which may last 5 to 12 hours. After this period the patient becomes more and more dyspneic, suffers from severe cough, and becomes cyanotic, as a result of the development of pulmonary edema which may end fatally in the course of several days. This type appears to be the most common, as illustrated by the reports of many.

b. The reversible type of nitrous fume poisoning is characterized by dyspnea, cyanosis, vomiting, vertigo, somnolence, a feeling of intoxication, fainting, loss of consciousness and methemoglobinemia. This group of patients does not develop pulmonary edema and if removed early enough from the exposure they may recover completely, but otherwise the poisoning may rapidly end fatally.

c. The shock type of nitrous fume poisoning immediately shows severe symptoms of asphyxiation, convulsions and respiratory arrest, death being presumably due to interference with the pulmonary circulation resulting in stasis of the blood vessels. This form appears to be exceptional and may result from sudden inhalations of high concentrations.

d. The combined type of nitrous fume poisoning immediately shows symptoms from the central nervous system, such as vertigo, somnolence, and staggering gait. There may be some cyanosis. After apparent recovery from this stage may be followed, after some

hours, by progressive dyspnea, marked cyanosis and pulmonary edema. The pulmonary edema may be complicated with pneumonia.

e. In regard to the behavior of the circulation in nitrous fume poisoning, the action of the heart may be influenced by way of the medullary centers, but as pulmonary edema develops and as the oxygenation of the blood becomes less effective, the heart has to perform a greater amount of work under less favorable conditions and this may lead to dilitation, especially of the right ventricle, which is not uncommon in such poisonings.

f. The number of red cells may be considerably increased, which is probably the result of blood thickening on account of the pulmonary edema, and is also associated with an increase in hemoglobin which may reach very high values.

(D) *Chronic Poisoning:*

(1) Sequelae of Chronic Exposure to Nitrous Fumes.

a. Irritation to the eyes, upper respiratory tract, ulceration of the mucous membranes of mouth, and larynx, the latter occasionally requiring intubation to prevent suffocation; deterioration of teeth and inflammatory reaction to the gums.

b. Chronic exposure to low concentrations may also lead to respiratory affections as chronic bronchitis and emphysema; may also affect the circulatory apparatus causing hypotonia and bradycardia.

(E) *Pathology:*

(1) The pathological changes in the upper respiratory tract may be characterized by hyperemia of larynx, trachea and bronchi.

(2) The lumen of the bronchi may be narrowed by peribronchial fibrosis and by organized plugs filling the lumen. In less severe cases, fibrinous flakes may swim in the edema fluid filling the bronchi as observed in phosgene poisoning.

(3) Bronchiolitis obliterans may be one of the late sequelae of nitrous oxide poisoning. Most authors agree that the gross pathologic picture is very similar to that of miliary tuberculosis, which also holds true for the X-ray pictures of such lungs. Clinical evidence may be negative in contrast to the pathologic and X-ray findings.

(4) Abdominal organs are usually more or less hyperemic, the blood vessels being dilated and engorged. Degenerative necrosis, regional thrombi and hemorrhages in the esophagus and duodenum may be explained on the basis of circulatory failure. With delayed death there may be considerable hemosiderosis of various organs.

(5) In the brain, hyperemia and multiple small hemorrhages, resulting in purpura are the most common findings.

(F) *Toxic Concentrations:*

(1) The maximal allowable concentration of nitrogen Dioxide in air for continued exposure has been suggested as 1.4 to 2.5, 10, and 40 parts per million by volume.

TOXICITY OF NITROGEN DIOXIDE FOR MAN (Lehmann and Hasegawa, 1913)

Concentration			
Calculated as HNO_3 mg/liter	Calculated as NO_2 mg/liter	Parts per million by volume	SYMPTOMS
			(Self experiments of Hasegawa)
0.16	0.12	64	Moderate irritation of larynx, respiration slightly increased and shallow.
.26	.19	100	Marked irritation of larynx, cough.
.54	.39	207	Very marked irritation of nose and larynx, cough, increased nasal secretion and lacrimation.
			(Lehmann's conclusion)
.1	.07	37	May be tolerated for several hours by many persons.
.2	.14	74	May be tolerated for ½ hour.
.3- .4	.22-.29	117-154	Dangerous with somewhat prolonged exposure.
.6-1.0	.44-.73	234-388	Rapidly increasing danger.

(G) *Sanitary Corps Officer:*

(1) Proper storage nitric acid.

(2) Exhaust facilities are properly installed and functioning in all operations which may result in the formation of nitrous fumes, such as nitrating operations, pickling of metals, etc.

(3) Instructions to operators relative to accidental spilling of nitric acid, i.e.: washing away with large quantities of water and *not* use sawdust and other organic materials. In case of accidental formation of nitrous fumes, the room should be evacuated without delay. It should be entered only by special personnel familiar with the potential dangers of nitrous fumes and provided with proper gas masks or open air helmets. This crew should provide adequate ventilation and remove the source of the toxic vapors.

(H) *Medical Control:*

(1) Preemployment placement examination should include X-ray of chest.

(2) Persons suffering from bronchitis, asthma or other pulmonary affections or from diseases of the heart, should be excluded from such operations.

(3) Periodic check every six (6) months with particular reference to teeth, respiratory and circulatory systems.

(4) Treatment

a. Removal from exposure as soon as possible and kept absolutely quiet for at least 24 hours, even if his condition does not appear alarming. Medical attention should be easily accessible.

b. Inhalation of mist of 5% sodium bicarbonate solution.

c. Administration of oxygen to increase the oxygenation of the blood as needed.

d. Artificial respiration should be restricted to very few and selected cases because forceful compression of the thorax may lead to very serious complications.

e. Pulmonary edema may be favorably influenced by venesection.

f. Morphine seems detrimental due to its depressant effect upon the respiratory center. Codeine and barbiturates will serve the purpose and are less harmful.

g. Cardiac stimulants may become necessary in case of imminent cardiac failure.

h. Following recovery from the acute effects of the poison, the patient should be watched carefully for some time because late pneumonias and bronchiolitis obliterans may develop during the following weeks.

SELECTED REFERENCES

Banham, H. A. L., Haldane, J. S., and Savage, T.: The Presence Post Mortem of Nitric-Oxide-Haemoglobin. Brit. Med. Jour., vol. 2, pp. 187-189, Aug. 1, 1925.

Both, B.: Fatal Poisoning with Nitrous Gases. Samm. v. Vergift., vol. 7 (Vergiftungsfalle), pp. 177-178, 1936.

Broch, O. J.: Poisoning by Oxides of Nitrogen. An Investigation of Cases at the Rjukan Saltpetre Factories. Norsk. Mag. f. Laegevidenskapan., vol. 98, pp. 370-397, 1937.

Carboneschi, Carlos, L.: Poisonous Gases. VIII. Nitrous Oxide and Nitrogen Dioxide. Semana med. (Buenos Aires) vol. 1, pp. 375-382, 1938.

A Clinical Study of Acute Cases of Poisoning by Oxides of Nitrogen and their Prevention: Med. du Trav., vol. 6, pp. 196-206, 1934.

Coltman, R. W.: Gases from Carbon Arcs. J. Ind. Hyg. Toxicol., vol. 20, pp. 289-296, 1938.

Cramer, G.: Pneumonitis Caused by Nitrogen Oxides. Arch. Gewerbepath. Gewerbehyg., vol. 9, pp. 1-12, 1938.

Freitag: Dangers of Nitrous Gases. Z. ges. Schiess- u. Sprengstoffw., vol. 33, pp. 15-16, 1938.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 12-16.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 134-136.

Holste, A.: Poisoning by Nitric Oxide in Human Subjects. Zentr. Gewerbehyg. Unfallverhut. vol. 23, pp. 183-186, 1936.

Kissinger, P.: Note on the Decree of the Reich's Minister of Labor on the Generation of Nitrous Oxide in Autogenous Welding. Aerztl. Sachverstand.-Ztg., vol. 43, p. 122, 1937.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 630, 657.

Koelbel: Another Fatal Gas Poisoning in Acetylene Welding in a Boiler. Arbeitsschutz, p. 21, 1938.

Kohn-Abrest, E.: The Determination of Nitrogen Peroxide Vapors in Air and its Usefulness in Industrial Hygiene. Chim. et indust., Special No., pp. 947-949, April 1928.

Krauland, W.: Fatal Accident from Nitrous Gas. Wien. klin, Wchnschr., vol. 52, pp. 493-495, 1939.

MacQuiddy, E. L., Tollman, J. Perry, LaTowsky, Leroy W., Bayliss, Milward: The Combustion Products of the Carbon Arc. J. Ind. Hyg. Toxicol., vol. 20, pp. 312-320, 1938.

McNally, W.: Toxicology. Industrial Medicine, Publishers Chicago, 1937, pp. 408-411.

Nordmann, M.: Fatal Nitrous Gas Poisoning in Welding in a Closed Boiler, Aerztl. Sachverst.-Ztg., vol. 43, pp. 145-148, 1937.

Occupation and Health International Labour Office Geneva, 1934. Vol. 2, pp. 355-365.

Poisoning with Nitric Acid Fumes: Lancet, vol. 2, p. 1218, Dec. 7, 1929.

Public Health Bulletin No. 271, The Aromatic Amino and Nitro Compounds, Their Toxicity and Potential Dangers.

Sander, Fritz: The Appearance of Nitrogen Oxides in Grog Gas Burners. Z. Hyg. Infektionskrankh., vol. 121, pp. 142-147, 1938.

Simonin, C., and Stauder, R.: Poisoning by Nitrous Fumes Given off When Certain Explosives are Used in Mines. Med. Du travail, vol. 2, pp. 343-347, 1930.

Vigdorchik, N. A., Andreeva, E. C., Matusevich, I. S., Nikulina, M. M., Frumina, L. M., and Striter, V. A.: The Symptomatology of Chronic Poisoning with Oxides of Nitrogen. J. Ind. Hyg. Toxicol., vol. 19, pp. 469-473, 1937.

Public Health Bulletin No. 272, The Toxicity and Potential Dangers of Nitrous Fumes.

PETROLEUM DISTILLATES

Chemical Formula and Synonyms:

(Petroleum ether) Benzine; bensinum; purificatum U.S.P.; canadol; light ligroin; petroleum naphtha.
(Pentane) C_5H_{12}, amyl hydride; isopentane; normal pentane.
(Heptane) C_7H_{16}, dipropylmethane; heptyl hydride; methyl hexane; normal heptane.
(Octane) C_8H_{18}.
(Gasoline) Petrol; motor spirit.
(Kerosene) Coal Oil.
(White spirits).

A. Principal Properties:

1. (Petroleum ether) A mixture of several of the lighter constituents of petroleum (cymogene, rhigoline and gasoline). Sp. Gravity 0.635 to 0.660; b. p. 40° to 70° C. Soluble in alcohol, ether, chloroform, benzene and fixed and volatile oils (except castor oil).
2. (Pentane) A colorless, mobile, inflammable liquid; pleasant odor. Sp. Gr. 0.630.
3. (Heptane) Volatile, colorless liquid; highly inflammable. Sp. Gr. 0.682.
4. (Octane) Colorless liquid; Sp. Gr. 0.706.
5. (Gasoline) Fractional distillate of petroleum with boiling range between 85° C. and 200° C.
6. (Kerosene) Kerosene oils are also known as burning oils, lamp oils, and illuminating oils.
7. (White Spirits) A term used in England for turpentine substitutes. Sp. Gr. 0.785.

B. Principal Industrial Uses:

1. (Petroleum ether) Solvent.
2. (Pentane) Anesthetic, artificial ice manufacture, filling low-temperature thermometers; lubricant for claude liquid air machine.
3. (Heptane) Anesthetic; solvent.
4. (Octane) Solvent; fuel.
5. (Gasoline) Fuel for internal combustion engines; solvent; cleansing clothing, etc.; paint mixing; rubber cements.
6. (Kerosene) Fuel; illumination.
7. (White Spirits) Turpentine substitutes.

C. Poisoning—Systemic:

1. Modes of entry.
   a. Chiefly inhalation of its vapors.
   b. Absorption through skin must be considered in certain types of poisoning.
2. Symptoms.
   a. Considering the possible diversity of composition of petroleum distillates, the variety of symptoms encountered through exposure to its vapors is not surprising. For this reason, the discussion of the sympoms of poisoning will be limited to a consideration of petroleum distillates in general.
   b. Acute: An acute anesthetic action sometimes resembling alcoholic inebriation. Headache, blurred vision, mental confusion, inability to do fine work, dizziness, nausea, abdominal pain and loss of consciousness are common symptoms. A striking feature of acute gasoline poisoning is the severe muscular jactitation occurring during the excitement stage. In some cases the movements are as violent as in an epileptic convulsion and are particularly marked during recovery in fresh air. Sequelae from severe non-fatal poisoning have been reported, coming on three or four months afterwards. Epilepsy, motor paralysis involving the pyrimidal tracts and retrobulbar neuritis have followed cases of acute poisoning.
   c. Chronic Poisoning: Symptoms of chronic petroleum distillate poisoning are not very characteristic and it is difficult to make such a diagnosis with certainty. The most typical symptoms are loss of weight, with periods of recovery almost to normal, pulse rate sometimes notably increased, blood picture a severe secondary anemia, skin irritation, mouth infection, diarrhea, mental depression, with slowed mentality.

3. Laboratory Findings.

   a. Blood.

   Chronic Exposure—Hemoglobin decreased; R.B.C. secondary anemia; color index sub-normal; erythrocytes punctuated red cells; total W.B.C. a moderate leucocytosis.

4. Toxic Concentrations.

   a. Five-tenths of a milligram per liter is the safety limit for calculating ventilation.

   b. The amount tolerated in air is 0.5 up to 2 mg. per liter of air.

   c. 10 to 20 mg. per liter may produce harmful effects.

   d. The toxicity of Benzine is increased when the temperature is raised.

D. **Sanitary Corps Officer:**

1. Supervision of workers exposed to petroleum distillates.
2. Workers should not be exposed to skin-wetting by petroleum distillates under any prolonged condition of employment; they should also be protected from unusual or protracted inhalation of the vapors.
3. Proper control methods and use of suitable masks when petroleum distillate exposures are present.

SELECTED REFERENCES

Duvoir. Occupational Disease Caused by the Handling of Hydrocarbons and Their Principal Derivatives. Ann. med. legale criminol police sci., vol. 8, pp. 453-501, 1928.

Flinn, Frederick B., A. B., Ph. D.: Some of the Potential Public Health Hazards from the Use of Ethyl Gasoline. Jour. Ind. Hyg., vol. 8, no. 2, pp. 51-66, Feb. 1926.

Frolow, N.: A Case of Mass Poisoning in the Rubber Factory of "Krassny Bogatyr." Gigiena Truda, no. 1, pp. 79-82, 1926.

Guensei, F.: Benzine Poisoning. Deutsch. Ztschr. f. allg. Heilk., vol. 146, pp. 15-19, 1938.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 181-186, 1934.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetraethyl Lead. Part I. Jour. Ind. Hyg., vol. 16, no. 2, pp. 100-128, March 1934.

Kidston, T. A.: Intoxication Following the Use of Coal Tar Paints. Med. Jour. Australia, vol. 1, pp. 188-189, Feb. 13, 1926.

Kintz, G. M.: Safety in the Petroleum Industry. Mines Mag., vol. 28, pp. 304-308, 1938.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924.

Machle, Willard, Scott, E. W., and Treon, Joseph: The Physiological Response to Isopropyl Ether and to a Mixture of Isopropyl Ether and Gasoline. Jour. Ind. Hyg., vol. 21, no. 3, pp. 72-96, March 1939.

McNally, W.: Toxicology,-Industrial Medicine, Publishers, Chicago, 1937.

Occupation and Health. International Labour Office, Geneva, 1934.

Ohio Department of Health, Petroleum Distillutes, 1940.

Pugliese, A.: Experimental Investigations of the Toxic Action of Benzine. Petroleum Ether and Toluene. Rend. ist. Lombardo, vol. 55, pp. 443-444, 1922.

St. George, A. V.: The Pathology of the Newer Commercial Solvents. Am. J. Clin. Pathol., vol. 7, pp. 69-77, Jan. 1937.

Schustrow, N., and Letawet, F. K.: Fatty Substances in Benzine Poisoning. Deutsch. Arch. f. Klin. Med., vol. 154, p. 180, March 1927.

Schustrow, N., and Salistowskaja, E.: Ibid. p. 277, Jour. Am. Med. Assn., vol. 86, p. 1254, April 17, 1926.

PHENOL: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Phenol) C_6H_5-OH, carbolic acid, hydroxybenzene, phenic acid, phenyl hydroxide, phenylic acid.
(Creosote) Creosote oil, liquid pitch oil.
(Cresol, ortho) CH_3-C_6H_4-OH, cresyl alcohol, ortho-cresylic acid, ortho-kresol, ortho-methylphenol, ortho-oxytoluene.
(Cresol, meta) CH_3-C_6H_4-OH, cresylic acid, meta-cresylic acid, meta-kresol, meta-methylphenol, meta-oxytoluene.
(Cresol, para) CH_3-C_6H_4-OH, para-cresylic acid, para-kresol, para-methylphenol, para-oxytoluene.
(Creolin) Cresoline, kresosolvin, sanatol.

A. Principal Properties:

1. Phenol
   a. White crystalline mass which turns pink or red if not perfectly pure or under influence of light; absorbs water from the air and liquefies; sharp burning taste; distinctive odor; strong, corrosive poison. When in weak solution it has a sweetish taste. Sp. Gr. 1.071 at 25/4° C., m.p. 42-3° C.; b.p. 181.4° C. Soluble in alcohol, water, ether, chloroform, glycerol, carbon disulfide, petrolatum, fixed or volatile oils and alkalis. Weight per liter of vapor, 3.92, gr.
2. Creosote
   a. Yellowish to dark green-brown, oily liquid; clear at 38° C. or higher, characteristic odor: poisonous. Frequently contains substantial amounts of naphthalene and anthracene. Sp. gr. 1.03 to 1.10 distilling range 200° to 400° C. Soluble in alcohol, benzol and toluol.
3. Cresol, ortho
   a. White crystals; phenol-like-odor; poisonous. Sp. gr. 1.050; m.p. 30.4° C.; b.p. 191° C. Soluble in alcohol, ether and chloroform; slightly soluble in water.
4. Cresol, meta
   a. Colorless to yellowish liquid; phenol-like odor; poisonous; Sp. gr. 1.04; m.p. 10.9° C.; b.p. 202° C. Soluble in alcohol, ether, and chloroform; slightly soluble in water.
5. Cresol, para
   a. Crystalline mass; phenol-like odor; poisonous; Sp. gr. 1.039; m.p. 36° C.; b.p. 202° C. Soluble in alcohol, ether, and chloroform; slightly soluble in water.
6. Creolin
   a. A disinfectant consisting of a cresol-rosin soap solution. It mixes in all proportions with absolute alcohol, chloroform, and ether, but forms a milky emulsion with water. It is considerably less toxic than phenol, although it is from two to four times stronger as a disinfectant. Phenol coefficient, without organic matter, 3.25; with organic matter, 2.52.

B. Principal Industrial Uses:

1. Phenol
   a. Disinfectant for sanitary, medical and surgical purposes; manufacture of picric acid, salicylic acid, phenacetin and various intermediates for the production of dyes; paint and varnish removers; synthetic resins and plastics; phenates; brewing (cleansing agent): explosives; synthesis of artificial tannins; disinfecting and germicidal paints; synthetic perfumes; pharmaceutical (germicide antiseptic, deodorizing preparations, germicidal soaps).
2. Creosote
   a. Wood preservative; disinfectants; thermometers.
3. Cresol, ortho
   a. Disinfectant; coumarin.
4. Cresol, meta
   a. Disinfectant; fumigating compositions; production of synthetic resins; photographic developer; nitrocresol explosives.
5. Cresol, para
   a. Disinfectant; fumigating compositions; cresotinic acid; dyestuffs.

6. Creolin
   a. Disinfectant.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation.
   b. Absorption through skin.
   c. Ingestion accidental.

2. Symptoms

Phenol

a. Phenol is noted for its powerful caustic effects upon the skin and mucous membranes. When applied to the skin it causes sensations of tingling and numbness and in the contrated form an eschar is formed that falls off in a few days leaving a brown stain. The anesthetic properties of phenol are well known in the practice of medicine. In minute amounts it may cause an eczema which may be brought about either by actual contact with phenol containing substances or by the presence of phenol vapors in the air. When taken internally it causes a burning pain and a corrosion of the tissues in the mouth, throat and stomach.

In addition to its local action phenol may produce marked changes in the central nervous system. In mild cases the chief symptoms are headache, dizziness and sometimes excitement with mild delirium. In severe cases unconsciousness intervenes, the pulse is weak and rapid and respiration irregular. Convulsions rarely occur and death results from failure of respiration.

Since phenol is a general protoplasmic poison which enters into combination with cell proteins it is not surprising to observe symptoms of blood degeneration. Emaciation, nephritis, jaundice and gangrene may be encountered under certain conditions. Bandages should not be applied to skin surfaces which have come in contact with phenol as this inhibits the evaporation of the phenol. Gangrene has been known to occur under these conditions.

Cresol

a. Cresol is a mixture of three isomeric cresols and is frequently sold under the name of tricresol. The symptoms are essentially the same as those listed under phenol and the toxicity is approximately of the same order. Cresol is sold in the form of a suspension in water with soap under the name of Lysol.

Creosote

a. Creosote is a complex mixture of phenols and their ethers obtained from wood tar. The term is also applied to a similar product obtained from the distillation of coal tar. The toxic properties are similar to those of phenols, the nature and severity depending upon the specific material under consideration. Epithelioma of the skin after prolonged exposure to creosote has been reported. It is believed that acridine may be regarded as the effective irritating principle in tar, creosote and pitch which sensitizes the skin to light.

D. Sanitary Corps Officer:

1. Proper storage and control measures.

SELECTED REFERENCES

Duvoir, M., Hazomann, R., Desoille, H., and Fallot, P.: Occupational Poisoning by Cresol. Bull. mem. soc. med. hop. Paris, vol. 54, pp. 106-11, 1938.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 169, 195, 199, 200, 234, 250.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, p. 163.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 205, 335, 350, 634 and 918.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 665, 805 and 809.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 552-553.

Ohio Department of Health, Phenol and Related Compounds, 1940.

PHOSGENE

Chemical Formula and Synonyms:

(O= C= Cl_2, carbonyl chloride.)

A. Principal Properties:

1. Colorless, volatile liquid; extremely poisonous; Sp. gr. 1.392; m. p. —126° C; b. p. 8.2° C. Very slightly soluble in water, very soluble in benzol and acetic acid. Weight per liter, 4.11 gr.

B. Principal Industrial Uses:

1. Lethal gas for warfare; bleaching sand for glass manufacture; chlorinating agent; dye manufacture (methyl violet).

C. Poisoning—Systemic:

1. Modes of Entry
   a. Inhalation.
2. Symptoms
   a. The toxic action of phosgene is primarily associated with its effect on the respiratory system. It may cause violent lung inflammation with edema, necrosis of lung tissue, emphysema, bronchitis, bronchietosis, disfunction of the heart and dyspnea. In moderate concentrations the immediate effects may be slight but due to the liberation of free hydrochloric acid in the deeper respiratory structures, congestion and edema may occur later. If this is not fatal, lobar pneumonia possibly with abscesses may result. Phosgene is formed by the decomposition of certain chlorinated hydrocarbons. This is an important fact in connection with the use of carbon tetrachlorido as a fire extinguisher or any industrial use in which this solvent may be decomposed. The increasing use of various types of chlorinated hydrocarbons in dry cleaning processes should carry with it a reminder of this hazard.
3. Pathology
   a. The chief pathologic effects of phosgene poisoning are manifested in the upper respiratory tract, the bronchi, pulmonary tissues, and the blood vessel walls. Edema, interstitial inflammation, stasis, thrombosis with embolus formation, and degenerative changes in the nerves were the predominant sequelae.
   b. The formation of hematin in the blood after phosgene poisoning is attributed to the action of the hydrochloric acid which is liberated by the hydrolysis of the phosgene.
4. Toxic Concentrations

	Parts of Phosgene per Million Parts of Air
Least detectable odor*	5.6
Least amount required to cause immediate irritation to the eyes*	4.0
Least amount required to cause immediate irritation to the throat*	3.1
Least amount required to cause coughing*	4.8
Maximum concentration allowable for prolonged exposure*	1.0
Dangerous for even short exposure*	25
Rapidly fatal	Over 25

D. Sanitary Corps Officer:

1. Occupations which offer contact with phosgene are:*
   a. Carbontetrachloride workers.
   b. Dye makers.
   c. Phosgene makers.

Achard, Lebanc, and Binet: The Blood in Poisoning by Carbon Oxychloride (Phosgene). Archives de medecine experimentale et d' anatomie pathologique, March, 1920.

Budelmann, G.: Clinical Aspects of Poisoning by Inhalation (Blue Cross, Green Cross, Yellow Cross Gas. Carbon Monoxide, Benzene). Med. Klin., vol. 34, pp. 1029-1032, 1064-1066, 1928.

Delepine, Sheridan, M. B., C. M., M. Sc.: Summary of Notes on Two Fatalities Due to Inhaling Phosgene ($COCl_2$). Jour. Ind. Hyg., vol. 4, no. 10, pp. 433-440, Feb., 1923.

D'Osvaldo, E.: Clinical Contribution on the Effect of Phosgene Gas. Ann. di ottal., vol. 56, p. 154, Feb. 1928.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 7-12.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 136-137.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 518, 576 and 630.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 418-421.

Mayer, H.: The Formation of Blood Pigments by Phosgene. Deutsch. med. Schnschr., vol. 54, pp. 1557-1558, 1928.

Neilsen, J. Behn: Behavior of Gas-Mask Charcoal Towards Phosgene and Chlorine. Z. ges. Schiess-Sprengstoffw., vol. 27, pp. 280-284, 1932.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 621-626.

Ohio Department of Health, Phosgene, 1940.

Watjen. Medical Society of Halle.: Pathologic-Anatomic Findings in War—Gas poisoning. Munchen. med. Wchnschr., vol. 80, p. 750, May 12, 1933.

Wohlwill, F.: Pathological Anatomy of the Phosgene Poisoning. Deutsch. med. Wchnschr., vol. 54, pp. 1553-1557, 1928.

PHOSPHORUS: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Phosphorus) P_4.
(Phosphine) PH_3, phosphoretted hydrogen, hydrogen phosphide, phosphuretted hydrogen.
(Phosphorus Trichloride) PCl_3, phosphorus chloride.
(Phosphorus Pentachloride) PCl_5, phosphoric chloride, phosphoric perchloride.

A. Principal Properties:

1. Phosphorus
   - *a.* Yellow. Light yellow, wax-like, semi-transparent, crystalline solid; luminous and phosphorescent in the dark; distinctive, disagreeable odor; exceedingly poisonous.
   - *b.* Red. A solid non-metallic element. Bright reddish-brown, odorless amorphous powder; non-poisonous.
   - *c.* Black Phosphorus (metallic phosphorus, Hittorf's phosphorus) is a denser allotropic form of yellow phosphorus, obtained by crystallizing the latter from molten lead. It is also obtained by heating red phosphorus in sealed tubes to 360° C. for a long time. Sp. gr. 2.32.

	(a)	*(b)*
Specific gravity	1.82 at 20° C.	2.20 at 20° C.
Melting point	44.1° C.	590° C. at 43 atm.
Boiling point	280° C.	Ignites in air at 725° C.

Flash point: (a) Spontaneously ignites in air. (b) Only ignites above 200° C. (a) Soluble in carbon disulfide, benzol and chloroform, slightly soluble in alcohol, ether and fixed oils. (b) Soluble in absolute alcohol, insoluble in carbon disulfide. (a) Wt. per liter of vapor, 5.16 gr.

2. Phosphine
   - *a.* Colorless; spontaneously inflammable gas; disagreeable, garlic-like odor; exceedingly poisonous. Sp. gr. 1.146 (A); liquid, sq. gr. 0.746 at 19° C. M. p. —132.5° C.; b. p. —85° C. Soluble in alcohol, ether, and cuprous chloride; slightly soluble in cold water; insoluble in hot water.
3. Phosphorus Trichloride
   - *a.* Clear, colorless fuming liquid; decomposes rapidly in moist air. Sp. gr. 1.574 at 20.814° C.; m. p. —111.8° C.; b. p. 75.95° C. at 760 mm. Soluble in ether, benzol, carbon disulfide and carbon tetrachloride; decomposed by water. Wt. per liter of vapor 5.71 gr.
4. Phosphorus Pentrachloride
   - *a.* Slightly yellow, crystalline mass; irritating odor; fuming in moist air; strong irritating effect on the eyes. Sp. gr. (solid) 1.6; (gas) 3.60 at 295° C. (A); m. p. (under pressure) 148° C. Ordinarily sublimes without melting, sublimes 160° C. Soluble in carbon disulfide; decomposed by water. Wt. per liter of vapor, 8.66 gr.

5. Important Compounds
   a. (Phosphorus) Phosphorus bolognian, oxychloride, pentachloride, sesquisulfide, trichloride; phosphotungstic acid, phosphate rock, phosphine, phosphomolybdic acid, phosphor bronzes, phosphoric acid; phosphoric acid, glacial; phosphoric acid, reverted.

B. Principal Industrial Uses:

1. Phosphorus
   a. Match industry. Now prohibited in most countries (not in Italy); manufacture of rat poison; phosphor-bronze; production of phosphorus pentachloride and other compounds; medicine (treatment of diseases of the osseus tissues).
   b. Match industry instead of poisonous, yellow phosphorus; organic synthesis.
2. Phosphine
   a. Organic preparations.
3. Phosphorus Trichloride
   a. Chlorinating agent; solvent for phosphorus; irridescent metallic deposits; manufacture of saccharin.
4. Phosphorus Pentachloride
   a. Chlorinating agent in organic chemistry; catalyst.

C. Poisoning—Systemic:

1. Modes of entry.
   a. Inhalation.
   b. Ingestion.
   c. Absorption through skin.
2. Symptoms
   a. (Phosphorus) Industrial poisoning by phosphorus formerly associated with the use of white phosphorus in the manufacture of matches, fortunately is a thing of the past due to the substitution of phosphorus sesquisulfide for the dangerous white phosphorus. Change in the bone structure, particularly of the jaw, is the outstanding effect of exposure to fumes of white phosphorus. These fumes soften any decayed teeth in the exposed individual, penetrate to the periosteum of the jaw bone and set up a partial necrosis. This leads inevitably to a suppurative inflammation because of the constant presence of pathogenic organisms. Abscesses form and eventually bone undergoes necrosis.
   The first symptom of industrial phosphorus poisoning is toothache which increases in severity and finally becomes excruciatingly painful. If the tooth is pulled and the worker returns to the phosphorus fumes before the wound is completely healed, the symptoms of suppurative inflammation rapidly develop. In the worst cases it has been necessary to remove all of both jaw bones and in certain cases the necrosis has extended to the orbit with loss of the eyes.
   The changes in the periosteum that result in the loss of the jaw bone may also occur in the other bones. Injuries to the bones in the legs of workers exposed to phosphorus fumes have resulted in necrosis and destruction of these bones with symptoms similar to those usually observed in the jaw bone.
   b. (Phosphine) The acute action of phosphine is said to resemble that of food poisoning. There is marked dyspnea, purgation, weakness, tremors, and finally convulsions and death. Its effects are apparently exerted largely through the central nervous system. Prolonged exposure to small amounts of phosphine is said to give rise to symptoms identical with those of phosphorus poisoning.
   The following table indicates the physiological response to various concentrations of phosphine:

Physiological Response to Various Concentrations of Hydrogen Phosphide.

	Parts of Hydrogen Phosphide per Million Parts of Air
Maximum amount that can be inhaled for 1 hour without serious result*	100 to 200
Dangerous in 30 minutes to 1 hour*	400 " 600
Rapidly fatal**	2000

*Kobert, R., Kompend. d. prak. Toxikologie, Stuttgart, 1912, 45.
**Rambousek, Industrial Poisoning, London, 1913, 191.

c. (Phosphorus Trichloride) Phosphorus trichloride is an irritant to the respiratory tract. Its vapors readily decompose in the presence of moisture with liberation of hydrochloric and phosphorus acids. This decomposition is more rapid than that of phosgene, and as a result, phosphorus trichloride is more irritating to the upper respiratory tract than phosgene. Its action is said to be a combination of gas such as hydrogen chloride which acts primarily in the upper respiratory tract and phosgene which acts primarily on the deeper respiratory structures.

d. (Phosphorus Pentachloride) The action of Phosphorus Pentachloride is similar to that of phosphorus Trichloride.

e. Skin irritation due to Phosphorus Sesquisulphide develops rapidly, first in the exposed parts, but soon becomes generalized. The eruption is erythematous primarily but rapidly becomes vesicular or pustular. Occasionally, small necrotic areas appear. The outbreak is accompanied by severe subjective sensations. The attack rapidly subsides. A severe conjunctivitis is often present. For the eruption a watery solution of potassium permanganate and sodium bicarbonate is indicated.

3. Pathology

a. Necrotizing effect upon the jaw and acute yellow atrophy of the liver.

D. Sanitary Corps Officer:

1. Phosphine poisoning may occur among acetylene welders, as acetylene contains approximately 0.04% phosphine and very small quantities of arseniuretted hydrogen. Adequate measures of ventilation, washing facilities, separate rooms for eating, transfer of phosphorus workers from time to time, and instructions to workers with regard to hazard.

E. Medical Control:

1. Pre-employment placement examination

a. Dental infections predispose workers to jaw necrosis when exposed to phosphorus and they should not be placed in that type of operation.

b. Periodic check of persons exposed to this hazard and dental check should be included.

c. Transfer of employees from time to time and especially after extraction of teeth.

SELECTED REFERENCES

Analysis of Factory Inspection Reports, 1920-1922, III. Phosphorus: Internat. Labour Rev., vol. 13, pp. 550-557, April 1926.

Atkinson, H. V.: The Treatment of Acute Phosphorus Poisoning. Jour. Lab. and Clin. Med., vol. 7, no. 3, pp. 148-150, Dec. 1921.

Bell, R. D., and Doisy, E. A.: Rapid Colorimetric Methods for Determination of Phosphorus in Urine and Blood. Jour. Biol. Chem., 44, no. 1, 55, Oct. 1920.

Bucharest Letter: Prohibition of the Use of Both White and Yellow Phosphorus for Matches. Jour. Am. Med. Assn., vol. 83, p. 371, Aug. 2, 1924.

Cirla, P.: The Phosphorus Content of Blood and of Bone in Various Types of Experimental Poisoning; Lead, Mercury, Chromium, Phosphorus, Strontium and Fluorine. Med. del Lav., vol. 28, pp. 44-54, 1937.

Engel, K.: Mode of Action of Phosphorus. Arch. f. exp. Path. u. Pharmakol. vol. 102, pp. 289-304, 1924.

Frie, M.: Matchbox Dermatitis. Med. Klin., no. 16, 1921.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 86-91.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 182-84.

Kawada, T., and Yamasaki, F.: Urine of Phosphorus-Poisoned Men. Jour. Biochem., Tokyo, vol. 21, pp. 423-425, 1935.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 526-534.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 84-88.

Maitland, C. T.: Phosphorus Poisoning in Match Factories in China. (Report to the Industrial Committee of the National Christian Council of China) China Jour. Science and Arts, Feb., vol. 3, p. 20, March 1925.

Mariconda, P.: Experimental Investigations on Chronic Poisoning with Phosphorus. Arch. farm. sper. vol. 39, p. 24, 1925.

Muller, W.: Toxicity of Phosphorus Smoke. Gasschutz u. Luftschutz, vol. 4, p. 326, 1934.

Nicholas, J., Gate, J., and Rousset, J.: Cutaneous Phosphoritis. A new External Cause of Dermatitis. Bull. Soc. franc. de dermat. et syph., pp. 210-212, March 1929.

Nicholas, J., Gate, J., and Rousset, J.: A New Occupational Dermatitis Seen in the Phosphorus Industry. Med. du travail, vol. 2, Compt. rend. du Congres, pp. 331-336, 1930.

Occupation and Health. International Labour Office, Geneva, 1944, pp. 628-635.

Ohio Department of Health, Phosphorus and its Compounds, 1940.

Oliver, T.: Phosphorus Poisoning in Match Factories. Jour. State Med., vol. 32, pp. 334-339, July 1924.

Rasch, C.: Match Box Dermatitis and Conjunctivitis. Ugoskrift for Laeger, vol. 83, no. 34, p. 1119, Aug. 25, 1921.

Salvioli, I., and Sacchetto, I.: Contribution to the Study of the So-Called Fatty Degeneration of the Liver in Inanition and in Phosphorus Poisoning. Il Lavoro, vol. 12, no. 8, pp. 229-230, Dec. 31, 1921.

Simonds, J. P.: The Mechanism of the Protective Action of Carbohydrate Diet in Phosphorus and Chloroform Poisoning. Arch. Int. Med., vol. 23, no. 3, pp. 362-379, Mar. 15, 1919.

Staemmler, M.: Investigations Concerning Diastatic Ferments in the Liver, Especially in Cases of Phosphorus Poisoning. Munchen. Med. Wchnschr., vol. 73, p. 627, April 9, 1926.

Wertham, F.: Central Nervous System in Acute Phosphorus Poisoning. Arch. Neurol. and Psychiat., vol. 28, p. 320, 1932.

Zangger, H.: Accidents and Dangers of Work Inside Closed Receptacles. Arch. f. Gewerbepath. u. Gewerbohyg., vol. 4, pp. 117-140, 1932.

RADIOACTIVE LUMINOUS COMPOUND

The standard medical control of employees exposed to Radioactive Luminous Compounds is essential to safeguard these employees against serious injury and even death.

A. Principal Properties:

1. The material known as Radioactive Luminous Compound or "Luminous Material" or "Radium Paint" is a mixture of phosphorescent zinc sulphides and radium, mesothorium, or similar radioactive substance.
2. Usually packed in small glass bottles holding one gram of the compound.

B. Principal Uses.

1. Mixed with an adhesive to form a "paint" just before application.
2. To make visible markings in total darkness.

C. Poisoning:

1. Modes of entry
   - *a.* Ingestion or inhalation of solid radioactive luminous compound.
   - *b.* Inhalation of radon liberated from compound into air.
   - *c.* Exposure of whole body to gamma radiation from compound.
2. Symptoms
   - *a.* No immediate effects.
   - *b.* Accumulation is a slow process.
   - *c.* Damage to skeletal system. Bone necrosis followed by secondary anemia and leucopenia.
   - *d.* Later effects—Bone necrosis and osteogenic sarcoma, aplastic anemia.
3. Laboratory
   - *a.* Anemia and leucopenia.
   - *b.* X-ray of bone revealing bone necrosis.
4. Toxic Concentrations
   - *a.* Any employee who shows a deposit of more than 0.1 microgram of radium, as revealed by the expired air test.
   - *b.* Atmosphere concentrations of work rooms shall not exceed 10^{11} curie per liter according to present knowledge.
   - *c.* The whole body exposure of the worker to gamma radiation shall not exceed 0.1 roentgen per working day.

D. Pre-employment Physical Requirements:

1. General—Personnel must be selected with care and only those employed who are naturally neat and careful.
2. Vision—Normal or correctible to normal.
3. None should be employed who have a history of anemia, tuberculosis or syphilis.
4. Dental—particular attention to teeth and jaws.
5. Special attention to the skeletal system.
6. Laboratory—x-ray of chest, Kahn Test, Urinalysis, total R.B.C., total W.B.C., Hg. determination, differential count.

E. Control Measures:

1. Medical
   - *a.* Occupational disease examination every six (6) months with particular attention to teeth, jaws and bones in general.
   - *b.* Blood picture.

2. Sanitary Corps Officer

   a. Monthly determination of Radon test of expired air after worker is away from the workroom for at least twelve hours.

   b. The Radon concentration of the atmosphere of workroom shall not exceed 10^{11} curie per liter.

   c. Determination of exposure to Gamma radiation.

F. Printed Instructions to Supervisors of Luminous Workers:

1. Personal Cleanliness.—Radioactive luminous compound must be treated as any other poisonou substance. Therefore, the worker must develop habits of extreme personal cleanliness in the workroom. The compound must not be spilled or scattered, and it must not come in contact with the hands or clothing to any appreciable extent. At the end of the working period, the hands shall be carefully washed with the solvent for the particular adhesive used. This shall be done in such a way as to remove all traces of compound. When mixed with adhesive the compound is not readily removed by soap and water. No edibles of any kind, including chewing gum, candy or beverages, shall be brought into the workroom, nor shall they be touched before removing all traces of compound from the hands. A convenient method of inspection to determine whether the hands and clothing are free of compound consists in viewing them in a darkroom by means of light from an argon bulb. The worker shall perform this inspection regularly under supervision, whenever leaving the workroom.

2. Neatness in the workroom.—The skill required for application of luminous compound demands neat and orderly methods of procedure. Therefore, a skillful worker may be expected to keep all utensils and equipment in a neat and clean condition at all times. Compound shall not be permitted to accumulate and all utensils shall be left clean at the end of each working period.

3. Tipping of Brushes.—In some cases very fine markings are coated with radioactive paint, requiring a fine tip on the brush. This should be achieved by the selection of a proper size and shape of brush and by manipulation of the brush in the container for the mixed paint. *At no time shall the brush be pointed by the lips or fingers.* It is essential that the adhesives contain solvents or a substance which are distasteful to prevent the habit of pointing brushes between the lips. Experience has shown that the latter practice has been largely responsible for many of the fatalities which have occurred in the past.

4. Supervision of Personnel.—Dial painters and others engaged in handling luminous compound shall be under constant and competent supervision to make sure that all recommended practices are strictly followed. It shall be the supervisor's duty to inspect utensils and equipment for neatness and cleanliness, and to examine the worker's hands at the end of working periods and after washing. This should be done under an argon bulb to see that all compound has been removed. Rules regarding bringing food, candy, chewing gum, or beverages into the workroom shall be strictly enforced. The supervisor shall also inspect, daily, rest rooms and lunchrooms (if available on the premises) to make certain that cleanliness is maintained and that no articles contaminated with luminous compound find their way into these rooms.

G. Disposition:

1. Any worker who reveals evidence of Radon absorption or storage in the bones by the Radon test, shall change his occupation immediately and be treated by decalcification therapy.

2. When the atmosphere concentration of Radon exceeds 10^{11} curie per liter, request immediate investigation as to the cause.

3. When Gamma Radiation exceeds 0.1 roentgen per working day, immediate investigation as to cause is necessary.

H. **Suggested Examination for Radioactive Luminous Compound Workers at Six Month Intervals:**

1. Name and Date
   - *a.* Past Exposure
   - *b.* Past Illness
   - *c.* Symptoms (Present)
   - *d.* Vision
   - *e.* Teeth and Gums
   - *f.* Throat
   - *g.* Skeletal System (X-ray of bone if necessary)
   - *h.* General Chest Examination
   - *i.* Total Red Blood Count
   - *j.* Total White Blood Count
   - *k.* Hemoglobin
   - *l.* Differential
2. Sanitary Corps Officer:
   - *a.* Radon of Expired Air
   - *b.* Radon of Atmosphere
3. Remarks:

SELECTED REFERENCES

U. S. Department of Commerce, National Bureau of Standards, Hand Book, H27, Safe Handling of Radioactive Luminous Compound, 2 May 1941.

U. S. Department of Commerce, National Bureau of Standards, Hand Book H38, Protection of Radium During Air Raids, 4 May 1942.

U. S. Department of Commerce, National Bureau of Standards, Handbook H23, Radium Protection, 25 August 1938.

SILICOSIS AND SIMILAR DUST DISEASES

The pneumoconioses represent a class or type of diseases of the lungs which develop slowly as a result of occupational exposure. The term "pneumoconiosis" may be applied to any pathological condition of the lung produced by the inhalation of dusts. It is common further to classify pneumoconiosis according to the chief constituents of the dust producing the condition; for example, silicosis produced by silica, asbestiosis produced by asbestos, siderosis produced by iron, anthracosis produced by carbon particles, etc. Since silicosis is the most important and control measures effective in preventing silicosis are applicable in the prevention of other forms of pneumoconiosis, this discussion will be limited chiefly to reporting the cause and prevention of Silicosis.

Definition of Silicosis: A disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes, and the development of miliary nudulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic X-ray findings.

A. Principal Properties:

1. It occurs in two forms, free and combined. The free silicas as a group are definite compounds in the form of SiO_2. The combined forms are silicates. Of the free silicas which occur in nature, quartz is the most common. Quartz is a hard mineral and chemically resistant to reagents. Other forms of free silica are Opal (SiO_2H_2O), amorphous hydrated form, tridymite, cristobalite, and siliceous glass, or vitreous silica.

B. Principal Industrial Uses:

1. Owing to the fact that the earth's crust contains so great an amount of silica, it is obvious that those occupations concerned with the driving of tunnels, development of highways, and mining are frequently associated with a silicosis hazard. Those industries that have to do with the processing and industrial use of mineral products, such as the smelting and refining of ores, the use of sand and gravel for structural purposes, the carving of stone, particularly granite, the manufacture and use of certain abrasives, and the processing of the various forms of free silica, expose workers to this hazard.

C. Pulmonary Effects:

1. Modes of entry
   a. Inhalation of free silica particles 10 microns and less in size.
2. Symptoms
   a. The subjective and objective signs of silicosis vary according to the rate of development and degree of pulmonary fibrosis, and when infection is present, according to the type, extent, and duration of the complicating pulmonary infection. From a medical point of view, there is no sharp line of demarcation as to stages of silicosis. However, by means of a description by stages, the progressive nature of the disease may be better illustrated.
   b. First Stage: Chest film indicates early nodular fibrosis, the individual may or may not exhibit such clinical evidence as slight shortness of breath upon exertion, and some cough. The general appearance is that of a healthy individual, and there is no appreciable decrease in capacity to perform usual duties.
   c. Second Stage: More advanced nodulation is shown by X-ray, and there may be some evidence of localized aggregation of nodules and pleural thickening. Definite shortness of breath upon exertion, cough more pronounced, chest movements sluggish, and expansion usually decreased. The individual may be able to continue at his usual job, although less effectively.
   d. Third Stage: Fibrous changes as shown by X-ray film are further accentuated. Nodules are larger and assume conglomerate form, showing large shadows corresponding to areas of dense fibrosis. Shortness of breath is marked and distressing upon slight exertion. Cough is increased, and may be dry, but is usually productive. Chest expansion is decreased even upon forced inspiration. The individual's capacity for work is seriously and permanently injured.
   e. The diagnosis is based upon historical data, including the complete occupational history, past and present medical history; clinical, laboratory and X-ray findings. Except by autopsy, silicosis cannot be diagnosed definitely until X-ray examination reveals characteristic shadows of both lung fields. Other evidence of complicating pulmonary disease should be included in the diagnosis.

3. Predisposing Causes
   a. Age and geographical in itself is no great factor.
   b. Previous exposure seems to have a definite influence in predisposing to silicosis.
   c. Role of Infection: Infection developing along the respiratory tract may be of importance. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis, bronchiolectasis, emphysema, asthma, and pleurisy all tend to decrease the ability of the lung to rid itself of foreign materials, through lessened lymphatic drainage and decreased power to force the bronchial secretions and foreign matter from the lungs.

4. Pathology
   a. Tissue reaction to dust: It has been shown that silica in solution or non-crystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibreblastic cells, while other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected.
   b. Size of Dust Particles: Since dust, to exert its harmful action, must enter the finer divisions of the lung, the particle size of the atmospheric dust bears a definite relationship to the injurious effect produced. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from ever reaching the finer divisions of the lung, and such as do are likely to be expelled with the bronchial secretions. The soluble silica plays a definite part in the production of the disease, and the size of the particle also affects the rate of solution, due to the fact that the smaller the particles, the greater the total surface area exposed to the action of solvents.

5. Prognosis
   a. In the main, the prospects for recovery for cases of Silicosis are not favorable. Even when removed from dust exposure, the disease often tends to progress and to be complicated with tuberculosis. If the individual can be removed from his dusty occupation before serious damage to the lungs has occurred, his chances of maintaining his working capacity are fair.

6. Standard of Dustiness
   a. In an investigation of the granite cutting plants conducted by the U. S. Public Health Service, most of the workers were found to be exposed to an averagge of about 60,000,000 particles of dust less than 10 microns in the greatest diameter per cu. ft. of air. The dust contained 70% silica, of which about 35% was in the form of quartz or free silica. Under such conditions, there was an almost universal occurrence of silicosis among those exposed twenty years or longer, and a large proportion of workers developed pulmonary tuberculosis. It was found in this investigation that little or no evidence of lung injury could be observed in workers exposed to a concentration of between 9,000,000 and 20,000,000 particles, so that this report suggests in broad limits a tentative concentration of air dustiness which can be tolerated over a period of years without serious consequence. Obviously such an estimated limit would be valueless where conditions are dissimilar. Most accepted permissible limit is 5 million particles per cu. ft. (size range 5 to 10 microns). While obviously dust containing 90% silica is more harmful than dust containing 60%, it is not certain that the relative harmfulness is one of strict arithmetical proportion. Another value of dust counts lies in their use as a measuring rule to indicate the relative efficiency of devices and the method adopted to suppress dust within an industry.

D. **Sanitary Corps Officer:**

1. Measures for control of dust hazard.
2. Maintenance of existing control measures.
3. Supervision of protective devices for workers exposed to dust hazard.

E. **Medical Control:**

1. Pre-employment placement examination should include X-ray of lungs. All persons who have chronic infections of the upper respiratory tract should not be exposed to dusty occupations.
2. Periodic Examinations
   a. A yearly complete physical examination which should include X-ray of lungs.

F. **Suggested Silica Periodic Check:**

1. Name, Address, Age, Race, Sex, Department, Age Began Work and Number of Years Worked.

2. Present:

   a. Specific Occupation
   b. Specific Industry
   c. Number Years In:
      (1) Dust
      (2) Non-Dust

3. Past:

   a. Specific Occupation
   b. Specific Industry
   c. Number Years In:
      (1) Dust
      (2) Non-Dust

4. Past Illness:

5. Present Illness:

   a. Cough
   b. Shortness of Breath
   c. Fatigue
   d. Others

6. Physical Examination:

7. Laboratory:

8. X-Ray

9. Remarks:

SELECTED REFERENCES

Riley and Dallavolle, Public Health Reports, vol. 54, No. 3, 3 March 1939, pp 352-359.

A Study of Quartz-Fusing operations with special Reference to the Measurement and Control of Silico Fumes.

Sayers and Jones, Public Health Reports, vol. 53, No. 33, 19 August 1928, pp 1453-1472.

Silicosis and Similar Dust Diseases.
Metropolitan Life Insurance Co., Industrial Health Section, Silicosis.

SULFURIC ACID

Chemical Formula and Synonyms:

(Sulfur Dioxide) SO_2 sulfurous acid, anhydride.
(Sulfur Trioxide) SO_3, sulfur trioxide, anhydride of sulfuric acid.
(Sulfuric Acid) H_2SO_4, dipping acid, oil of vitriol.

A. **Principal Properties:**

1. Sulfur Dioxide
   a. Colorless gas or liquid; suffocating odor; corrosive poison.
   Gas: Sp. gr. 2.264 (air) liquid: Sp. gr. 1.434; m.p. —72.7° C.; b.p. —10° C.
   b. Soluble in water.
2. Sulfur Trioxide
   a. Colorless prisms. Sp. gr. gas: 2.75 (A); liquid 1.923; m.p. 16.83° C.; b.p. 44.6° C.
   b. Soluble in sulfuric acid.
3. Sulfuric Acid
   a. Strongly corrosive, dense, oily liquid; colorless to dark brown, depending on purity. Ordinary commercial (not pure) acid dissolves all metals, including platinum, although the latter dissolves very slowly. Concentrated acid (65%) when cold attacks iron, aluminum, copper and lead slowly. Heating the concentrated acid increases its action on metals, except when the boiling point of the acid is passed. Dilute acid dissolves aluminum, chromium, cobalt, copper, iron, manganese, nickel, zinc, and other metals, particularly if heated. It does not dissolve lead or mercury, and has very little action on high-silica iron. Sulfuric acid rapidly disintegrates wood, rubber, textiles and organic materials in general.
   b. Sp. gr. 1.8342; m.p. 10.49 C.; b.p. 210° to 338° C. Soluble in water in all proportions with evolution of heat.

B. **Principal Industrial Uses:**

1. Sulfur Dioxide
   a. Chemicals (sulfuric acid, ozone from hydrogen peroxide; Hargreave's process of salt cake manuture, sulfites, hydrosulfites and meta-bisulfites of potassium and sodium, alum from shale, recovery of volatile substances); intermediates; bleaching straw, feathers, silk, basketware, sponges, oils, sugar juices, flour and foods; preservative for beer, wine, and meats; restoring the yellow color of the new grain to old barley and oats; cellulose and paper industries; artificial ice industry; disinfecting and fumigating; fire extinguisher in mine fires; tanning; field mouse destruction; agricultural fumigant; extraction of bituminous matters in lignite coal.
2. Sulfur Trioxide
   a. Used to prepare sulfuric acid by the catalytic process.
3. Sulfuric Acid
   a. Chemicals (manufacture of various organic and inorganic chemicals, particularly sulfates and acids); fertilizers; petroleum refining; explosives; leather industry; metallurgy (pickling iron and steel; precious metal refining; electroplating; cleaning copper and silver; metallurgy of copper, iron, magnesium, cobalt and nickel); electric batteries, rubber reclaiming; textile industry; rayon; sulfonating oils; synthetic perfumes; ceramics; manufacture of fatty acids; neutralizing alkaline reactions of fermenting liquors; removal of lime used to soften and dehair hide scraps used in the manufacture of glue and gelatin; drying agent; mineral colors; pyroxylin plastics; wax purification; sugar industry; tar washing; parchment paper; fungicide; weed destroyer; exterminator; photographic processes; process engraving and lithography; analytical reagent.

C. **Poisoning—Systemic:**

1. Modes of entry
   a. Sulfur Dioxide—Inhalation of the gas.
   b. Sulfur Trioxide—Inhalation of the gas.
   c. Sulfuric Acid—Ingestion, local corrosive action on the skin, and inhalation as a mist may be considered as possible means of industrial poisoning.

2. Symptoms

a. (Sulfur Dioxide) Sulfur Dioxide has an irritating effect upon the membranes of the respiratory tract. Acute poisoning by this gas is rare since it causes a violent reflex which acts as a defense against lethal concentrations, provided the man can escape quickly enough. Although sulfur dioxide is almost irrespirable to those unaccustomed to it, a decided degree of tolerance may be established to it after prolonged exposure. Haggard states that this tolerance is only apparent and is due to a chronic inflammation of the upper air passages which produces a tenacious mucus and deadens the protective reflex mechanism. The individual hazard is thereby increased since the gas can penetrate the deeper respiratory structure and may cause edema of the lungs.

Kehoe and his associates studied the effect of prolonged exposure to sulfur dioxide on 100 workmen exposed to the gas in the course of their employment. They found a significantly higher incidence of chronic, slight naso-pharyngitis and an alteration in the sense of smell and of taste. The men did not seem more susceptible to colds, but the duration of the colds averaged two times longer than the control group. Other symptoms were: a high rate of abnormal urinary acidity, dyspnea on exertion, increased fatigue, and sluggish or hyper active reflexes.

The following table indicates the symptoms produced by various concentrations of sulfur dioxide:

Physiological response to various concentrations of SO_2

	Parts of SO_2 per Million parts of air
Least detectable odor	3 to 5
Least amount causing irritation to the eyes	20
Least amount causing immediate irritation to the throat	8 to 12
Least amount causing coughing	20
Maximum concentration allowable for prolonged exposure	10
Maximum concentration allowable for short exposure, one-half to one hour	50 to 100
Dangerous for even short exposure	400 to 500

b. (Sulfur Trioxide) Sulfur Trioxide is rarely encountered as a pure gas since this oxide has a strong affinity for water vapor. The white fumes seen when sulfuric acid is heated is a mist formed by the recombination of sulfur trioxide with water vapor to form sulfuric acid. The physiological effects of sulfur trioxide would therefore be the same as those produced by the inhalation of sulfuric acid mist.

c. (Sulfuric Acid) The inhalation of the fumes or mist of sulfuric acid cause irritation of the respiratory tract. This irritant action is immediate and any prolonged symptoms which might occur are a result of the corrosive action of sulfuric acid on the membranes of the respiratory tract. From an industrial point of view sulfuric acid is more significant as an accident hazard than as a health hazard. Many severe and painful burns have resulted from the careless handling of concentrated sulfuric acid.

Ingestion of sulfuric acid is usually suicidal or accidental and the severity of the intoxication depends upon the concentration and amount of acid ingested. Severe corrosive effects and grave symptoms are produced almost immediately. There is an intense thirst with great difficulty in swallowing, extreme tenderness of the abdomen, restlessness and collapse. With the retching, vomiting, and destruction of tissue the patient becomes weaker and death may occur from shock.

D. Medical Control:

1. Pre-employment—Those with chronic bronchial trouble, as pulmonary disease or sinus disease, should not be placed where this exposure exists.

E. Sanitary Corps Officer:

1. Supervision of control measures.

SELECTED REFERENCES

Betz, C. E., Holden, J. H., and Handy, J. O.: Sulfur Dioxide in Air of Industrial Community. Indust. and Engin. Chem., vol. 25, pp. 774-776, July 1933.

Capron, P. C.: An Automatic Apparatus for the Determination of Small Quantities of Sulfur Dioxide in Air. Bull. soc. chim. Belg., vol. 41, pp. 588-596, 1932.

Chumanov, S. M., and Aksel' rod, M. B.: A Simplified Colorimetric Determination of Sulfur Dioxide in the Air. J. Applied Chem. (U.S.S.R.), vol. 11, p. 270, 1938.

Colrat: Ocular Lesions Peculiar to the Artificial Silk Industry. Arch. d' opht., vol. 46, p. 416, July 1929.

Coste, J. H., and Courtier, G. B.: Sulfuric Acid as a Disperse Phase in Town Air. Trans. Farady Soc., vol. 32, pp. 1198-1201, Aug. 1936.

Finkelstein, D. N.: Rapid Determination of Sulfur Dioxide in Air. J. Applied Chem. (U. S. S. R.), vol. 9, pp. 1347, 1936.

Giffin, S. W., and Skinner, W. W.: Small Amounts of Sulfur Dioxide in The Atmosphere. I. Improved Method for Determination of Sulfur Dioxide When Present in Low Concentrations in Air. Indust. and Engin. Chem., vol. 24, pp. 862-867, 1932.

Gurevich, V. G.: Determination of Small Concentrations of Sulfur Dioxide and Hydrogen Sulfide Present Together in Air. Jour. Russ. Phys. Chem. Soc., vol. 62, pp. 111-119, 1930.

Gurevich, V. C., and Vendt, V. P.: Determination of Small Concentrations of Sulfur Dioxide and Hydrogen Sulfide in Air. II. J. Gen. Chem. (U. S. S. R.), vol. 6, pp. 962-971, 1936.

Haggard, Howard, W.: Action of Irritant Gases Upon the Respiratory Tract. Jour. Ind. Hygiene, vol. 5, no. 10, pp. 390-398, Feb. 1924.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 3-6.

Hecker, I. W.: Hydrogen-Ion Concentration of the Saliva of Picklers (Metal Cleaning). Arch. f. Hyg., vol. 5, pp. 255-262, Feb. 1934.

Henderson, Y., and Haggard, H.: Noxious Gases. The Chemical Catalog Co., Inc., Publishers, New York, 1927, pp. 87, 127-128, 130.

Karol, S.: Results of the Investigations in the Pollution of Air with Sulfur Dioxide in the Zinc Industry Region of Silesia. Med. Doswiadczalna Spoleczna, vol. 15, pp. 296-326, 1932.

Kennon, B. R., M. D.: Report of a Case of Injury to the Skin and Eyes by Liquid Sulfur Dioxide. Jour. Hygiene, vol. 9, no. 11, p. 486-487, Nov. 1927.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 571, 661, 662.

McCord, C. P.: Household Mechanical Refrigeration with Special Reference to the Toxicity of the Refrigerants Utilized: Report 2 of the Committee on Poisonous Gases of the American Medical Association. Jour. Am. Med. Assn., vol. 94, pp. 1932-1933, June 7, 1933.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 60-65, 439-444.

Meiter, E. G., and Traubert, C. E.: Sulfur Dioxide in the Air at the Pittsburg Experiment Station of the U. S. Bureau of Mines. U. S. Bur. Mines, Rep. Investigations, Serial no. 3005, pp. 5, June 1930.

Methods for the Detection of Toxic Gases in Industry. No. 3. Sulfur Dioxide. Dept. of Scientific and Industrial Research. H. M. Stationery Office, London, 1939.

Nikulina, M. M., Hetmann, Z., and Margolina, N. M.: Morphologic and Biochemical Changes in the Blood in Acute Sulfur Dioxide Poisoning. Sbornik Rabot po prefessionalnoy patologie i profkonsultazie (Repts. of Invest.), Leningrad Inst. for Invest. of Indust. Diseases, vol. 8, pp. 89-93, 1934.

Occupation and Health. International Labour Office, Geneva, 1934, pp. 976-979, 988-994.

Ohio Department of Health, Sulfuric Acid, 1940.

Schade, Carlisle: Sulfur Dioxide in Pittsburgh Air. Jour. Ind. Hygiene, vol. 15, no. 3, pp. 150-153, May 1933.

Schwarz, L., and Deckert, W.: Concerning Dust Containing Sulfuric Acid. Zentralbl. f. Gewerbehyg., N. S., vol. 3, pp. 314-315, Nov. 1926.

Setterstrom, Carl and Zimmerman, P. W.: Sulfur Dioxide Content of Air at Boyce Institute. Contrib. Boyce Thompson Inst., vol. 9, pp. 171-178, 1938.

Simferopol, L.: Sulfur Dioxide as an Injurious Impurity in the Room Air of a Fruit Preserving Factory and Measures for Its Removal. Gigiena Truda i Tekhnika Bezopasnosti, no. 5, pp. 90-92, 1934.

Smith, M. C.: Hazards of Sulfur Dioxide and Caustic Soda (in Water Works Plants). Committee Report. J. Am. Water Works Assoc., vol. 31, pp. 489-501, 1939.

Smith, Richard Brooke, and Frii, V. S. T.: Portable Motor-Driven Impinger Unit for Determination of Sulfur Dioxide. Jour. Ind. Hygiene, vol. 13, no. 10, pp. 338-342, Dec. 1931.

Tin-Plate Manufacture: Travelers Standard, vol. 12, pp. 105-110, May 1924.

Thomas, M. D., and Cross, R. J.: Automatic Apparatus for the Determination of Sulfur Dioxide in Air. Indust. and Engin. Chem., vol. 20, pp. 645-651, June 1928.

Tweddel, F.: Immunity to Tuberculosis Among Workers in Sulfur Dioxide. Med. Rec., vol. 98, no. 8, pp. 310-312, Aug. 21, 1920.

Ugnyackev, N.: Rapid Method for Determining Sulfur Dioxide in the Presence of Oxides of Nitrogen. Novosti Tekhniki, no. 10, p. 46, 1938.

Williams, C. L.: Sulfur Dioxide for the Fumigation of Ships. U. S. Pub. Health Repts., vol. 49, pp. 89-90, Jan. 19, 1934.

Williams, C. L.: Use of Liquid Sulfur Dioxide as a Fumigant of Ships. U. S. Pub. Health Repts., vol. 49, pp. 192-208, Feb. 9, 1934.

ZINC: RELATED COMPOUNDS

Chemical Formula and Synonyms:

(Zinc) Zn, blue powder.
(Brass) Alloy containing copper and zinc.
(Zinc Oxide) ZnO, Chinese white; zinc white; flowers of zinc.

A. Principal Properties:

1. Important Compounds
   a. (Zinc) Zinc acetate, bichromate, bromide, carbonate, chloride, cyanide, ethyl, fluoride, iodide, nitrate, oleate, oxide, phosphate, sulfate, sulfide.
2. Zinc
   a. Shining, white metal; bluish-gray luster; or gray powder. Sp. gr. 7.142; m. p. 419° C; b. p. 907° C. Soluble in acids and alkalis; insoluble in water.
3. Brass
   a. A copper zinc alloy of varying compositions. It also usually contains some lead or tin (sometimes both) and small percentages of other metals. Properties depend on the composition.
4. Zinc Oxide.
   a. Amorphous, white powder; absorbs carbon dioxide from the air. Sp. gr. 5.78; m. p. greater than 1800° C. Soluble in acids; insoluble in water and alcohol.

B. Principal Industrial Uses:

1. Zinc
   a. Slab Zinc. Galvanizing and electroplating: Wire, sheets, culverts, hardware, structural shapes, etc. Manufacture of non-ferrous alloys: Brass, nickel, manganese, bronze, tombec imitation gold. Manufacture of rolled zinc.
   b. Zinc castings. Slush castings, battery zincs; electroplating anodes.
   c. Rolled Zinc. Automotive equipment: Running board moldings, dome lamp rims, hub caps, escutcheon plates, scuff plates, curtain frames, drip and body moulding, tire valve nuts, gasoline tank caps, auto body lining. Electrical apparatus: Fuses, anodes, meter cases, insulator caps, ground plates, dry battery caps. Household Furnishings: Linoleum bindings, fruit jar caps, ice bag caps, laundry tags, wash boards. Building materials: Roofing, siding, leaders and conductors, gutters, flashings, shingles, fences, weather strips, corner beads, art glass strips, stair treads, pipe covering bands; glazier points, window bolt guards. Railroad car linings. Printing: Engravers' plates, lithographers' plates. Miscellaneous: Cable wrappings, eyelets, addressing machine plates, buttons, grammets, pin tubes, etched name plates, shoe lace tips, ice box drains and linings, foil, fence bands, templates, collapsible tube clips, embossed numbers and tags, gaskets, washing machine parts, casket ends, box linings, stencils, shoe nails, perforated screens, ornamental fittings, signs, organ pipes, hull plates, boiler plates.
   d. Zinc Dust. Chemicals (zinc salts, reducing agent in making sodium hydrosulfite for the reduction of vat colors); dyes (reduction of indigo blue); intermediates (reduction of nitro-benzene in the production of aniline, various synthesis); galvanizing by the Cowper-Cowles process; deoxidizing agent for bronzes; purification of fats; purifying agent in the treatment of solutions of zinc sulfate prior to electrolysis; manufacture of lithopone; bleaching bone glues; analytical reagent.
   e. Mossy Zinc. Stripping, photographic solution, sooth cleaners, coloring face brick, photographic chemical manufacture.
   f. Zinc Wire. Metal spraying.
2. Brass
   a. Plumbing fixtures, bearings, corrosion resistant alloys and other metallurgical applications.
3. Zinc Oxide
   a. Paint pigment; zinc salts; compounding ingredient in rubber manufacture; ceramic glazes; matches; linoleum (pigment); dental cements; pharmaceuticals (ointments and dusting powder for skin diseases); cosmetics, zinc soaps, opaque glass, white printing inks, candles, celluloid, textile printing (resist), white glue and gelatin.

C. Poisoning—Systemic:

1. Modes of entry
   a. Inhalation.
   b. Ingestion.

2. Symptoms
   a. Industrial poisoning by zinc is chiefly concerned with the inhalation of zinc oxide fumes. Inhalation of a sufficient amount of these fumes gives rise to a condition popularly known as "brass founders' ague," "zinc chills," and "metal fume fever." After a sufficient exposure, which depends upon the individual susceptibility, there may be a slight irritation of throat. The main symptoms, however, occur later, usually several hours after the victim has left the environment where the fumes prevailed. The attack resembles "malaria chills" and may last for several hours with ordinary febrile symptoms such as lassitude, headache, nausea, muscle cramps and joint pains, and constricting sensations over the lungs.
   b. Lehmann attributed the symptoms to a protein poison. The zinc oxide fumes kill certain cells lining the alveoli of the lungs and the resorption of the product of these cells gives rise to an attack similar to that produced by the injection of a foreign protein. Lehmann, however, was unable to explain why it was not possible to produce "metal fume fever" by insufflation of ordinary zinc oxide powder. Drinker showed that freshly formed oxide particles, such as occur in fumes of heated zinc, are not yet agglomerated and pass easily through the air passages into the alveoli of the lungs. It is evident, therefore, that metal fume fever is never caused except by the freshly formed fumes of heated metal.
   c. The ingestion of zinc compounds such as zinc sulfate, zinc chloride, etc., does not have important industrial significance. Soluble zinc salts are sometimes employed as emetics and locally as astringents and antiseptics. Zinc stearate powder is an ingredient in some dusting powders. Fatal pneumonia has resulted from the inhalation of this compound.
   d. Industrial health hazards may also prevail which are attributable to certain impurities rather than zinc itself. Arsenic, lead, and cadmium, all of which are more toxic than zinc, are frequently associated with it. Arsine poisoning is frequently caused by the treatment of zinc galvanized materials with acid.

D. Sanitary Corps Officer:

1. Control measures where metal fumes are being formed.

E. Medical Control:

1. Health hazard examinations for those who are exposed to metal fumes, once a year or oftener if exposures are severe.

SELECTED REFERENCES

Artur: Note on Some Cases of Poisoning by Zinc Fumes. Arch. de Med. et Pharm., vol. 199, pp. 547-559, Nov. 1929.

Batchelor, Roger P., M. D., Fehnel, J. William, B. S., Thomson, Robert M., and Drinker, Katherine R., M. D.: A Clinical and Laboratory Investigation of the Effect of Metallic Zinc, of Zinc Oxide, and of Zinc Sulphide upon the Health of Workmen. Jour. Ind. Hygiene, vol. 8, no. 8, pp. 322-362, August 1926.

Breyer, F. G.: Some Conclusions on Commercial and Experimental Methods of Collecting Dust and Fume in the Zinc and Blast Furnace Industries. Informal address before the Portland Cement Assn., Jour. Am. Soc. of Heating and Ventilating Eng., vol. 28, p. 288, 1922.

Burstein, A.: Brassfounder's Aguo. Gigiena Truda, no. 7, pp. 17-41, 1925.

Chrometzka, F.: Zinc Vapor Poisoning (Metal Fume Fever), Clinical Observations and Therapy. Deutsch. Arch. f. Klin. Med., vol. 179, pp. 569-576, 1937.

Drinker, Philip, B. S., Ch. E.: Certain Aspects of the Problem of Zinc Toxicity. Jour. Ind. Hygiene, vol. 4, no. 4, pp. 177-197, August 1922.

Drinker, Katherine R., M. D., and Collier, Ellen S., A. B.: The Significance of Zinc in the Living Organism. Jour. Ind. Hygiene, vol. 8, no. 6, pp. 257-269, June 1926.

Drinker, Katherine R., M. D., and Drinker, Philip, Ch. E.: Metal Fume Fever: V. Results of the Inhalation by Animals of Zinc and Magnesium Oxide Fumes. Jour. Ind. Hygiene, vol. 10, no. 2, pp. 56-70, Feb. 1928.

Drinker, K. R., Fehnel, J. W., and Marsh, M.: The Normal Excretion of Zinc in the Urine and Feces of Man. Jour. Biol. Chem., vol. 72, pp. 375-383, March 1927.

Drinker, Philip, Thomson, Robert M., and Finn, Jane L.: Metal Fume Fever: II. Resistance Acquired by Inhalation of Zinc Oxide on Two Successive Days. Jour. Ind. Hygiene, vol. 9, no. 3, pp. 98-105, March 1927.

Drinker, K. R., Thompson, P. K., and Marsh, M.: An Investigation of the Effect of Long-Continued Ingestion of Zinc in the Form of Zinc Oxide, by Cats and Dogs, Together with Observations upon the Excretion and the Storage of Zinc. Am. Jour. Physiol., vol. 80, pp. 31-64, March 1927.

Drinker, K. R., Thompson, P. K., and Marsh, M.: An Investigation of the Effect upon Rats of Long-Continued Ingestion of Zinc Compounds with Especial Reference to the Relation of Zinc Excretion to Zinc Intake. Am. Jour. Physiol., vol. 81, pp. 284-306, July 1927.

Drinker, Philip, Thomson, Robert M., and Finn, Jane L.: Metal Fume Fever: IV. Threshold Doses of Zinc Oxide, Preventive Measures, and the Chronic Effects of Repeated Exposures. Jour. Ind. Hygiene, vol. 9, no. 8, pp. 331-345, August 1927.

Engelsmann, R.: Lead and Zinc in Acetylene Workers Engaged in Wrecking. Klin. Wchnschr., vol. 2, pp. 1884-1887, Oct. 8, 1923.

Engelsmann, R.: Two Cases of Zinc Poisoning. Deutsch. Med. Wchnschr., vol. 48, no. 15, pp. 488-489, April 14, 1922.

Fairhall, Lawrence T., A. M., Ph. D.: The Estimation of Zinc in Biologic Material. Jour. Ind. Hygiene, vol. 8, no. 4, pp. 165-176, April 1926.

Fairhall, L. T., and Hoyt, L. H.: Excretion of Zinc in Health and Disease. Jour. Clin. Investigation, vol. 7, p. 537, Oct. 20, 1929.

Greenbaum, S. W.: Hygiene of Brass Foundries. New York City Dept. Health, Month. Bull., vol. 13, pp. 199-200, Sept. 1923.

Guelmann, J.: Metal Founders' Fever in Russia, Arch. f. Hyg., vol. 95, pp. 331-338, 1925.

Hamilton, A.: Industrial Toxicology. Harper and Brothers, New York, 1934, pp. 102-104.

Hargreaves, R.: The Vapor Pressure of Zinc in Brasses. J. Inst. Metals 64, Advance Copy no. 823, 10 pp. 1939.

Heller, V. G., and Burke, A. D.: Toxicity of Zinc. Jour. Biol. Chem., vol. 74, p. 85, July 1927.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, p. 331-334.

Koelsch, F.: Metal Fume Fever. Jour. Ind. Hygiene, vol. 5, no. 3, pp. 87-91, July 1923.

Lutz, Robert E., Ph. D.: The Determination of Small Amounts of Zinc in Materials of Organic Nature: A Microchemical Method Based on the Fluorescence of Zinc Salts with Urobilin. Jour. Ind. Hygiene, vol. 7, no. 6, pp. 273-290, June 1925.

Lutz, Robert E., Ph. D.: The Normal Occurrence of Zinc in Biologic Materials: A Review of the Literature, and a Study of the Normal Distribution of Zinc in the Rat, Cat, and Man. Jour. Ind. Hygiene, vol. 8, no. 4, pp. 177-207, April 1926.

McCord, C. P., Friedlander, A., Brown, W. E., and Minister, D. K.: An Occupational Disease Among Zinc Workers. Arch. Int. Med., vol. 37, pp. 641-659, May 1926.

McCord, Carey, and Kilker, C. H.: Zinc Chloride Poisoning. Report of Outbreak Among Workers in a Wood Preserving Industry. Jour. Am. Med. Assn., vol 76, no. 7, pp. 442-443, Feb. 12, 1921.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, p. 294.

Meyer, Walter: Preventing Occupational Disease During the Manufacture of Zinc Oxide. Ferben-Ztg., vol. 43, pp. 1197-1198, 1938.

Mills, C. M.: Joplin Zinc: Industrial Conditions in the Worlds Greatest Zinc Center. Survey, vol. 45, no. 19, pp. 657-666, Feb. 5, 1921.

Molfine, Francesco: Fever Produced by Breathing Metallic Vapors (in Workers in Marine Industry). (Occupational Chronic Intoxication by Zinc.) Rass. Med. Applicata Lavoro Ind., vol. 9, pp. 374-379, 1938.

Molfino, Francesco: Zinc Poisoning in Electrical and Autogenic Welding Employed in Naval Repair and Construction. Rass. Med. Applicato Lavoro Ind., vol. 8, no. 341-344, 1937.

Nuck, D., Remy, E., and Holtzmann, F.: Zinc Dust in Industry: Its Action on the Health of the Workers and Its Accumulation in the Organs. Reichsarbeitsble. (8), vol. 3, p. 92, 1929.

Occupation and Health, International Labour Office, Geneva, 1934, pp. 1277-1294.

Ohio, Department of Health, Zinc and Its Compounds.

Ray, Priyadaranjan, and Dutt, Nihar K.: Quinaldic Acid as Analytical Reagent. III. Determination of Zinc in the Presence of Copper, Silver and Mercury. Z. Anal. Chem., vol. 115, pp. 265-268, 1939.

Sturgis, Cyrus C., B. S., M. D., Drinker, Philip, B. S., Ch. E. and Thomson, Robert M.: Metal Fume Fever: I. Clinical Observations on the Effect of the Experimental Inhalation of Zinc Oxide by Two Apparently Normal Persons. Jour. Ind. Hygiene, vol. 9, no. 3, pp. 88-97, March 1927.

Thompson, Phebe K., M. D.: The Recovery of Small Amounts of Zinc from Biologic Material Ashed by the Incineration Process. Jour. Ind. Hygiene, vol. 7, no. 8, pp. 358-370, Aug. 1925.

Turner, J. A., and Thompson, L. R.: Health Hazards of Brass Foundries: I. Field Investigations of the Health Hazards of the Brass-Foundry Industry, II. Laboratory Studies Relating to the Pathology of Brass Foundry—Men's Ague. U. S. Pub. Health Bull. no. 157, pp. 75, Aug. 1925.

Zinc: Analysis of Factory Inspection Reports, 1920-1922, III. Internat. Labour Rev., vol. 13, pp. 546-549, April 1926.